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ARCHIVES OF OPHTHALMOLOGY.

CONCERNING THE VALUE OF THE BLOOD COUNT IN THE DIAGNOSIS OF SYMPATHETIC OPHTHALMIA.

BY S. R. GIFFORD, M.D., 1ST LIEUT., M.R.C.

SINCE Gradle, Ormonde, Browning, and Price-Jones emphasized an increase of lymphocytes in the blood of cases of sympathetic ophthalmia there has been some discussion as to the constancy with which this sign is present. In view of this disagreement, it seems that the report of careful differential counts on 9 cases of true sympathetic ophthalmia, 6 cases of recent perforating wounds of the globe, and 11 cases of prolonged or repeated non-traumatic inflammation of one eye, would not be without some value. It was intended to include repeated counts in a number of cases of chronic infection outside the eye, but circumstances have made this impossible, and it is hoped this data may be reported at a later time. The counts were made through the kindness of Doctor Harold Gifford on his private patients during the last two years.

Literature. Gradle (1 and 2) reported this increase in mononuclears in 6 cases of iridocyclitis after perforating injury, where there was danger of sympathetic ophthalmia. In one, the other eye had been affected and examination of the enucleated eye showed the changes of sympathetic ophthalmia. In the others, the condition of the second eye is not noted, so apparently no sympathetic ophthalmia developed. In all he found the mononuclears increased in some to over 40%. This

increase dropped to normal soon after enucleation of the injured eye. In 11 cases of perforating injury with quick healing, he found no such change, nor in 12 cases of non-traumatic iridocyclitis. The increase was most notable in the small lymphocytes. In a later report (3), he describes similar findings in 15 more cases of iridocyclitis after perforating wound. He mentions here finding cases of perforating wounds with uneventful healing which showed mononucleosis. He explains this change in both classes of cases according to Elschnig's anaphylactic theory, as due to absorption of uveal pigment sensitizing the organism to that pigment and producing the mononucleosis observed in sensitized animals. In the cases which show no inflammation, he assumes that there is slight absorption of antigen, but not enough to sensitize the organism, or else occurring in an organism not susceptible to such immunization.

Ormonde (4 and 5) and Browning and Price-Jones (6) at about the same time reported, in 9 cases of S. O. already developed, findings similar to Gradle's, except that the increase was most notable in the large mononuclears. They found that the count approached normal after salvarsan was given. Browning later (7) reports on 17 cases treated by salvarsan because of the similarity of the blood picture to that in protozoal diseases (1912). In 1913–14 (8) Browning again reports favorably on such treatment and notes that the typical blood picture develops before any signs of irritation in the second eye. But he also mentions 5 cases of S. O. with a normal count. He describes one which was normal for 3 to 4 weeks, counts being done twice a week, after which the second eye became inflamed and the mononuclears increased to 34%. He notes that other complicating conditions may lower the count, as in a case of pernicious anæmia and S. O. with normal differential count. He says his later counts, checked by Price-Jones, have not shown so high a mononucleosis, though some change is fairly constant.

Elschnig (9) had noted the occurrence of mononucleosis, but says it is simply one proof that the organism is reacting abnormally to the irritation of trauma or infection. The picture is varying after perforating wounds; either lymphocytes or polymorphs may dominate the picture, or it may be normal.

He says it cannot be maintained that mononucleosis is the specific reaction to the cause of sympathetic ophthalmia.

In the same way, Guilbert (10), speaking of the mononuclears in the exudate and in the tissues of enucleated cases, denies that this tells anything as to the cause of the uveitis; it merely shows a chronic inflammation. The more chronic the process, he finds, the more the lymphocytes preponderate.

Franke, in 1912 (11), reported counts in 51 cases of various kinds, made in an attempt to see if mononucleosis occurred in ocular conditions other than sympathetic ophthalmia. Of 21 old perforating wounds of the sclera followed by inflammation but quiet for some time when the count was made, 13 had 30% mononuclears and 2 more had 29%. Of 3 cases with some inflammation remaining, but not affecting the other eye, all had lymphocytosis. Six of ten perforating wounds of the cornea had lymphocytosis. Of 7 cases where enucleation had been done at first after perforating wound, he found mononucleosis in four. Two cases of contusion of the globe showed mononucleosis. He further describes a case of Mannhardt's and one of his own, both of whom showed a normal count some time after injury but required enucleation later because of inflammation of the second eye. He concludes that, since mononucleosis is present in so many cases where no S. O. occurs and absent in some where it does occur, its presence or absence is of no prognostic or diagnostic value in relation to sympathetic ophthalmia. He does not mention increase of the large mononuclears, but speaks of the mononucleosis as a lymphocytosis. He and Hack (12) refer to this condition in some further cases, and think it occurs especially in neurasthenic people.

Sattler (13) in discussing Franke's first paper says he has found the sign of value in diagnosing sympathetic ophthalmia.

E. A. Neumann (14) in a Berlin thesis, 1912, reports examinations in a large number of conditions. I was unable to find the original paper, but from reviews, it appears that these include 85 cases of recent injury and operative trauma with normal healing, suppuration after injury, traumatic iridocyclitis where S. O. was suspected; and tubercular infection, glaucoma, and non-perforating contusions. In each group, he

found lymphocytosis more or less frequently. He concludes that it has no prognostic or diagnostic value after injury.

From Fuchs's clinic A. Purtscher and E. Koller in 1912 (15) report results more in agreement with Gradle. In 9 cases of true sympathetic ophthalmia, 4 of which showed the characteristic changes at autopsy, lymphocytosis was found in all but one, averaging about 35%. The increase was mainly in lymphocytes the large mononuclears averaging 5%. In seven cases of traumatic iridocyclitis without S. O., the highest percentage of mononuclears was 27%, average 20%. They conclude that the sign is of diagnostic value. They mention that Wolfrum found an increase of lymphocytes in two cases of fresh S. O. and also note that Guilbert found this increase, but found no difference between the count in traumatic iridocyclitis and other cases of uveitis.

Of late the tendency in this country and in England seems to be towards acceptance of Gradle's or Browning's views. Thus Brownlie (16) advises blood counts at intervals between removal of a foreign body and recovery, to forestall S. O., but he gives no report of counts made. Pyle (17) also mentions it as important in diagnosis, but gives no particulars. Zentmayer (18), however, refers incidentally to a normal blood count in a case of beginning S. O. O. C. Hudson (19) reports the same in a case of developed S. O., the count being made by Browning. Treacher Collins (20) mentions one of his cases which had a normal blood count. Siegrist (21) found 40.5% mononuclears in a case, but six months later, after use of salvarsan when the eye was quiet, this count had risen to 47%.

METHOD OF STUDY

Where possible three white counts and differentials were made in each case, at intervals of 3 or more days. At least 200 cells were counted, unless otherwise noted. One cause of past disagreement has been the confusion between different observers as to what constitutes a large mononuclear. Gradle (2) has called attention to this, saying that the difference has been largely a personal matter and that the total mononucleosis is the important thing. Since I found a rather larger percentage of large mononuclears in nearly all counts than is considered

normal, it may be said that in this work any cell with a fairly clear, well-defined cytoplasm extending for more than two microns around the nucleus was considered a large mononuclear. Most were at least two to three times the size of a red cell, but some were included not much larger than a red cell, where the amount of clear cytoplasm seemed to put them plainly out of the class of lymphocytes.

Most of the cases were put on large doses of sodium salicylate, and it occurred to me that this drug might be a factor in producing a mononucleosis, as some other drugs, pilocarpine, etc., are known to do. Therefore in four perforating wound cases, counts were made before starting the drug, and at intervals after it was started. Four guinea pigs and four rabbits were given sodium salicylate both by mouth and by intravenous injection, the doses corresponding roughly to one grain per pound of their respective body weights, in a day. In two of the guinea pigs this was continued for 13 days, in two more for 6 days, in the rabbits for shorter periods. Counts were made before the drug was started, just after the first dose, at intervals of 2 or 3 days during its administration, and 3 days after it was stopped.

FINDINGS

Effect of Sodium Salicylate on the Blood Count of the 4 human cases (10, 11, 12, 13, included in Table B): two showed an increase in total mononuclears, one a slight increase followed by a drop to normal while the drug was being given, and one a distinct decrease. Realizing that the changing pathological conditions and the change from home to hospital life would render any such results of little value in judging the effect of the drug alone, it was hoped experiments on normal animals would be of more value. The blood picture in these was found to vary greatly and not constantly in any one direction. In both rabbits and guinea pigs the mononuclears always made up 40 to 80% of the total amount and their number bore no relation to the doses of sodium salicylate.

TABLE A.

NINE CASES RECEIVED AFTER THE OUTBREAK OF SYMPATHETIC OPHTHALMIA.

History	Date Counted	W. B. C.	L. M.	S. M.	Trans.	Total Monos.
1. James —— Seen 4 months after removal of R. E. for injury. L. E., vision at first shadows. L. E. shows iridocyclitis. Slowly became quiet under treatment with finally almost no light perception. No change while being counted.	3/14/18	10,400	32.5	9	3	44.5
	3/24/18	10,200	30.5	10.5	3.5	44.5
	3/28/18	18,020	17.5	11	3.5	32
	Average	12,870	26.8	10.2	3.4	40.4
2. Alvin —— Treated for S. O. 1 year before and now operated for seclusio pupillæ. No stirring up of inflammation by operation. Vision at first shadows. Final vision not recorded.	3/10/18	16,900	19	10.5	1	30.5
	3/17/18	15,200	15.8	13.5	1.7	28.9
	3/19/18	10,200	17	10	1.5	28.5
	Average	14,100	16.8	11.3	1.4	29.5
3. Mr. T—— Seen two months after injury to L. E. R. E. inflamed for 2 days. Vision of R. E., 20/10, L. E. 20/200. Scarlet fever when first seen and counted. R. E. responded slowly and improved only when atophan was given. Finally vision 20/30, and quiet at last count. L. E. always quiet.	3/24/17	17,800	18	8.5	.0	26.5
	3/26/17	24,800	23.5	4	2.5	30
	4/22/17	8,016	26.5	16	1	43.5
	Average	16,872	33.3	9.5	1.2	33.3
4. Mr. C—— R. E. perforated by iron, May 11. June 10, L. E. flared up. Seen June 17, and R. E. enucleated. Long course, with many deposits on Descemet's membrane, but vision 20/20. Papillitis recurring when last seen. Still under treatment. Secs. of R. E. showed changes of S. O.	6/17/17	10,440	15	2.5	15	18
	6/22/17	7,940	27.5	18	2	47.5
	6/24/17	6,440	32	22	3.5	57.5
	12/9/17	5,600	23.5	6.5	2	32
	Average	7,605	24.6	12.2	2	38.8

History	Date Counted	W. B. C.	L. M.	S. M.	Trans.	Total Monos.
5. Susie L—— Had S. O. 8 or 9 years ago. Recurrence with influenza. Examination of globe shows changes of S. O.	5/6/16	not taken	23.5	1	7.6	32.1
6. Mr. H—— 5 weeks before seen struck by nail in R. E. Vision of R. E.: fingers at 6 ft.; L. E. V. 20/20. R. E. enucleated followed by improvement of symptoms at once. Counts 7 and 18 days after nucleation when eye was quiet with opacities on anterior lens capsule. Final vision 20/20 L. E.	12/22/17 12/23/17 Average	6,200 6,800 6,500	18.7 10 14.3	26.5 8 47.2	3.6 5 4.3	48.8 23 35.9
7. Mrs. L. —— Seen December 20, 1915, 2 years after injury of L. E. R. E. shows acute iridocyclitis. L. E. blind. V. R. E. fingers at 2 ft. First count 3 days after arrival, while R. E. was suffering acute inflammation. At second count still irritated. Later rise of tension. Vision down to 2/200. Doubtful if true S.O.	12/23/15 1/3/16 Average	9,400 not recorded 9,400	36.4 7 18.7	4.2 12.5 8.3	.4 .5 .4	35 20 27.4
8. J—— Seen September 14, 1917. R. E. hurt 6 months ago and blind. L. E. shows iridocyclitis and descemetitis. Vision L. E. 20/30. Enucleated R. E. Final vision L. E. 20/40. L. E. quiet when counts taken; sections of R. E. showed changes of S. O.	10/26/07 10/27/07 Average	7,800 7,990 7,895	37.5 30 33.7	13.5 19 16.2	3 3 3	54 52 53
9. John S—— L. E. injured by knife 15 yrs. before and R. E. began to fail 4 weeks after this. L. E. removed 5 yrs. before. Now vision of R. E., fingers at 1 foot. Seclusio pupillæ. Eye quiet. Seen only February 25, 1918.	2/25/18	6,900	9	9	2	20

APPENDIX—TABLE A.

THREE CASES OF S. O. FOUND IN THE LITERATURE (IN CONNECTION WITH THE USE OF SALVARSAN) IN WHICH BLOOD COUNTS WERE RECORDED.

History	Case of	W. B. C.	L. M.	S. M.	Trans.	Total Monos.
Perforating wound of one eye. Inflammation of second eye. Enucleation of first eye. Count before enucleation.	W. Zentmayer	14,000	6.8	8.4		15.2
Inflammation started in second eye after injury to first. Count by Dr. Browning, before salvarsan was given.	A. C. Hudson	not given	9	not given		
Case of true S. O. with repeated exacerbations of uveitis in second eye. 1st count before salvarsan. 2nd count after salvarsan, when eye was quiet.	Siegrist 1/27/12 6/21/12 Average	 10,500 7,087 8,793	 3 2 2.5	 32.5 41 36.7	 5 4 4.5	 40.5 47 43.7

TABLE B.

SEVEN CASES OF PERFORATING INJURY WITHOUT INFLAMMATION OF SECOND EYE. ALL BUT ONE COUNTED SOON AFTER INJURY.

History	Date	W. B. C.	L. M.	S. M.	Trans.	Total Monos.
10. C—— Injury of R. E. seen soon after on same day. Put on large doses of sodium salicylate.	3/18/17	8,720	9.5	17	3.5	30
	3/19/17	7,620	16	15	1	32
	3/22/17	6,660	21	19	5.5	45.5
	Average	7,666	15.5	17	3.3	35.8
11. K—— 3/27/7. Perforating wound covered by flap and sodium salicylate started.	3/27/17	10,880	16	5.5	2.5	24
	3/29/17	9,532	19	11.5	1	31.5
	3/31/17	9,820	15	3	1	29
	4/1/17	5,900	12.5	8	4.5	25
	4/6/17	9,040	15.5	4	4.5	24
	Average	9,019	15.6	8.4	2.7	26.7
12. Mr. Y—— 3/29/17. Piece of stick in right eye. Removed this day and sodium salicylate started.	3/29/17	10,350	7	7.5	3	17.5
	3/31/17	12,020	8.5	4	1.5	14
	4/2/17	7,560	22.5	12	2	36.5
	4/6/17	8,690	14.5	8.5	2	25
	4/18/17	6,640	10.5	31.3	4.6	46.4
	Average	9,042	12.6	12.6	2.4	27.6

TABLE B.—*Continued*

History	Date	W. B. C.	L. M.	S. M.	Trans.	Total Monos.
13. Mr. G—— Cornea wounded by stick 2 days ago. Piece of stick removed and sodium salicylate started.	3/31/17 4/2/17 4/6/17 Average	8,350 13,020 11,340 10,900	13.5 17.5 12 14.3	18 8 3 9.6	2 5 1.5 2.8	33.5 30.5 16.5 26.7
14. Donald C—— Perforating wound of eye 3 weeks ago in child of 3. No inflammation of 2d eye. Injured eye showed some uveitis.	1/15/18 1/29/18 Average	14,600 20,140 15,370	6.5 16.5 11.5	5.5 8.5 7	3.5 3.5 3.5	15.5 28.5 22
15. B—— Old perforation of cornea healed after some inflammation, under sodium salicylate with vision 20/15.	2/20/18	9,440	17.5	5	3	25.5
16. Joe C—— Perforating wound of cornea R. E. Seen same day. No inflammation of second eye developed.	2/25/18	11,100	18.5	6.5	2.5	27.5

TABLE C

ELEVEN CASES OF VARIOUS CHRONIC INFLAMMATORY CONDITIONS

History	Date	W. B. C.	L. M.	S. M.	Trans.	Total Monos.
17. C—— Iritis for some months. Is 50 to 60 years old.	5/9/17	5,400	16.5	13	.5	30
18. K—— Glaucoma with iritis for some months. Count made some days after paracentesis. Final enucleation showed changes of glaucoma, no changes of S. O.	6/25/17	8,480	27.5	10.5	3	41.1
19. M—— Specific iritis treated by sodium salicylate some weeks before and after entrance.	3/7/17	14,860	11	15.5	5	31.5
20. M—— 4/2/17. Old iridocyclitis. Getting 120 grains sodium salicylate a day.	4/2/17 4/16/17 4/18/17 Average	12,860 6,640 6,920 8,960	20.5 13.5 8.5 14.2	4.5 6.7 2 10.7	4.5 3 5 4.2	29.5 23.2 39.5 29.1
21. R—— Old iridocyclitis getting sodium salicylate.	4/3/17	9,100	19	22	2.5	43.5

TABLE C—*Continued*

History	Date	W. B. C.	L. M.	S. M.	Trans.	Total Monos.
22. R—— R. E. lost by accident 7 years ago. Old irritable stump remains. Now cataract of L. E. First count before any treatment. Enucleation of old stump. 2nd count 2 days later. Cataract operation on L. E. with good result, some weeks later.	12/7/17 12/11/17 1/ 7/18 Average	13,400 10,200 ? 11,800	14.1 14.5 15 14.5	10.5 8.5 11.5 10.2	1 .5 1 .8	25.5 23.5 7.5 25.5
23. A—— Perforating wound of R. E. 2 months before. L. E. shows slight irritation. Vision about O. K. Referred to local doctor with advice to keep R. E. under watch. L. E. quiet.	1/7/18	6,400	18.5	8.5	2	29
24. B—— Old wound with abscess of cornea. Count made after long stay in hospital on sodium salicylate with healing.	1/30/18	8,700	15.5	13.5	7	36
25. H—— Had vernal catarrh this summer and for some years. Now quiet. No eosinophiles in secretion.	1/5/18	5,200	25.5	16	4.5	46
26. M—— Old obstinate uveitis of unknown origin. Resistant to all treatment.	11/20/17 11/21/17 Average	7,200 7,000 7,100	18.5 15 16.7	8 16.5 12.5	5.5 2.5 4	32 34 33
27. Isador W—— Old vernal catarrh. Eyes now look quiet but still itch.	11/23/17	7,000	17	24	.5	41.5

TABLE D

NORMAL CASE SHOWING MONONUCLEOSIS

History	Date	W. B. C.	L. M.	S. M.	Trans.	Total Monos.
Author's own blood.	5/9/18	6,400	14.5	32.5	5	52
	5/11/18	7,340	32	19.5	3	54.5
	5/18/18	10,680	23	17	2.5	42.5
	Average	8,140	23.2	23	3.5	49.7

SUMMARY

Group A. This includes 9 cases in which sympathetic ophthalmia had broken out in the second eye. The clinical diagnosis was made by H. Gifford, and in 3 cases sections of the enucleated eye showed the typical changes of sympathetic ophthalmia. In one case (Case 7) included in the table, the inflammation may not have been purely a sympathetic one. Of these, seven were counted during or shortly after the active inflammation. All of these, except the one doubtful case, had an increase in mononuclears above the normal. The highest count was 53%, the lowest 33.3%. All showed the increase, especially in the large mononuclears, the highest being 33.7% and the lowest 14.3%. On the two cases seen some time after the process had become perfectly quiet, one showed 29.5% of mononuclears (Ormonde calls 30% pathological) with an average of 16.8% large mononuclears. The other, on whom only one count was made, had only 20% mononuclears, 9% being large monos. In no case did the count change materially under treatment, or after enucleation, though in 5 cases the eye became fairly quiet, with good vision, before the last count was taken. Case 3 was complicated by scarlet fever, which caused much variation in the counts.

It was found generally that a leucocytosis of 12,000 or over made results variable and unreliable. In Group A, average total mononuclears was 34.9%; average large mononuclears 22.8%.

Of the three cases picked up from the literature, in which counts were made, 2 showed no increase in mononuclears which could be called pathological. The third had an average of 43.7% mononuclears, with only 2.5% large monos. and this mononucleosis was increased after the eye became quiet under salvarsan. In all Group A cases the inflammation had existed after the active process had set in in the second eye, and none earlier than two months after the injury, so that in all the process was a fairly old one.

Group B. In this group, on the other hand, acute processes are concerned. In all but two of the seven cases, counts were made within two days of the injury. In only one case did the average total mononuclear count exceed 30% (Case 10, 35.8%).

The lowest was 22%, the highest 35.8%. Large mononuclears were considerably increased in most, relative to the generally accepted normal. The highest was 18.5, the lowest 12.6, average 15.7. These figures refer to averages of several counts. In several individual counts, a mononucleosis, much above the normal, was found, as 45.5%, 46.4%, 36.5%, in three cases. If these had been taken alone and the count had been considered of importance in the fate of the injured eye, it might have been sacrificed unnecessarily, as all of these did well with no symptoms in the other eye. In only one of these was there any material decrease in mononuclears under treatment. Average total mononuclears, 25.9%.

Group C. Eleven cases of long standing affections of the eye, 6 of some form of uveitis, 2 of vernal catarrh, included because of the mononucleosis shown, one of old irritable stump left after a perforating wound which showed on microscopical examination no change suggesting S. O., one of long standing abscess of the cornea, one of suspected S. O., with very slight irritation of the second eye. It is notable that this last case was one of the few to show no marked mononucleosis (29%), though his large mononuclears were 18.5%. Only three of these cases showed an average of mononuclears under 30% and of these 3, two had 29%. The largest was 46%, lowest 25.5%, and average 35.1%. Four were over 40%. Large mononuclears were increased in all—highest 25.5%, lowest 14.2%, average 17.9%.

Group D includes only 3 counts made on myself within a period of ten days, and show that an apparently normal person may have a high mononucleosis. Average mononuclears 49.7%. Large mononuclears 23.2%. At other times counts on myself have been normal.

CONCLUSIONS

1. An increase of mononuclears was found to be fairly constantly present in cases of sympathetic ophthalmia. Average total mononuclears 34.9%.

2. This high count often persisted for a long time, even in perfectly quiet eyes, but in two cases was found reduced to normal after the eye had been quiet for years.

3. It was not found to be constant in fresh perforating

wounds which did not develop S. O., average 25.9%. It was sometimes found in these cases, however, in isolated counts.

4. A similar increase was found to be just as constant in other chronic affections, such as non-traumatic uveitis, abscess of the cornea, vernal catarrh, etc., as in S.O., average 35.1% (slightly higher than in Group A).

5. The increase in both cases was most marked in the large mononuclears, but the lymphocytes were also often increased. The personal factor must be considered here.

6. Large doses of sodium salicylate were not found to affect the count constantly in animals or men.

These results agree with Gradle and most of the above authors in finding the mononuclears increased in S. O. They disagree, however, with Gradle, Purtscher, and Koller, and with Browning in not finding the count change to normal immediately after enucleation, and in finding it persist in quiet eyes. One reason for this disagreement may lie in the fact that many of Gradle's cases had no actual inflammation of the second eye, and, as Elschnig says, might never have developed S. O. When the first eye was enucleated in these cases, the entire seat of inflammation was gone, whereas in the present series inflammation had already begun in the second eye, and persisted for a time after enucleation, some slight degree probably persisting even in eyes that appeared quiet. Browning, however, found the count to drop after enucleation even in cases with actual inflammation of the second eye. And it seems only natural that this should occur in many cases, though it did not appear notably in this series, when the source of inflammation in the first eye, whether of anaphylactic, protozoal, or infectious origin is removed. The more important point of disagreement between these findings and those of the above observers is in finding a similar count just as constant in cases of old uveitis and other chronic conditions. In this they agree with the larger series of comparative cases of Franke and Neumann and with Guilbert's experience.

7. It is the opinion of the author, therefore, that the mononucleosis is not specific for S. O., but is found as a reaction of the body to a chronic inflammatory process in the eye, of whatever cause. It is true, as Browning points out, that an increase in mononuclears occurs in protozoal and spirochætal dis-

eases, as malaria and syphilis, and, as Gradle says, that it occurs in sensitized animals and persons. But it is also true that, as any infection proceeds from the acute to the chronic stage, the primary leucocytosis, which principally involved the polymorphs, is replaced by a slighter leucocytosis, in which the proportion of mononuclears becomes increasingly greater. The mechanism by which this occurs is unknown, but apparently the mononuclears appear after exhaustion of the polymorph reaction has taken place. In view of our present uncertainty as to the course of S. O., this seems as reasonable an explanation of the mononucleosis which does occur in the disease, as any other.

In view, then, of the frequency with which it occurs in other conditions, and the occurrence of cases of true S. O. in which it is absent, it would seem very doubtful if the sign could be considered of any importance in determining the presence or absence of sympathetic ophthalmia, or the likelihood of its occurring in any case.

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REPORT OF A CASE OF BILATERAL PAPILLŒDEMA DUE TO EMPYEMA OF SPHENOID AND ETHMOID SINUSES. OPERATION AND RECOVERY.[1]

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THE patient, aged thirty-four, came to the writer's clinic at the New York Eye and Ear Infirmary for the first time on December 1, 1917, complaining of severe headache, which had forced him to give up his work two weeks before. He claimed the headaches were worse when he stood on his feet. He did not have the triad symptoms which generally accompany intracranial increase of pressure. There was no vomiting or vertigo. V. O. D. 20/20; V. O. S. 20/20. His blood pressure was: systolic 200, diastolic 110. His fields for form and color were normal except there was a slight enlargement of the blind spot. On examining the fundus of each eye I found that the patient had a marked case of papillœdema, with many hemorrhages. The optic disk in right eye could best be seen +9. Left eye could be seen best with +8, which is equal to about 3*mm* above the plane of choroid. The œdema and hyperemia were so great that the entire papilla, the physiologic cup, and the borders of the disk, including the scleral and pigment rings, had all disappeared and the whole picture presented the appearance of a solid mound. You could not see the vessels bending over the edge of the disks. They were lost in the excessive œdema of the surrounding retina. The engorgement manifested itself also in the enormous distention of the retinal veins. The arteries were smaller than normal. There were many hemorrhages on the swollen papilla and surrounding retina.

The patient was ordered into the Infirmary at once. Rest

[1] Patient was shown at the Ophthalmological Section of New York Academy of Medicine on March 18, 1918.

in bed, milk diet, and magnesium sulphate in the mornings. His urine showed a moderate amount of albumin, a few hyaline and granular casts, and a specific gravity of 1020. Wassermann was negative. Examination of teeth and tonsils was also negative. Patient gave history of having had a cold for some time and having had a discharge from the nose.

X-ray plate showed cloudy ethmoids, cloudy sphenoids, cloudy frontals on left side, and cloudy antrum of Highmore on the right.

While he was in hospital getting a rest cure for his kidney condition, his vision declined to 20/70 O. U. On December 16th, Dr. S. L. Craig operated upon and drained his ethmoid and sphenoid sinuses for me. There was some improvement following this operation, but his headaches still continued and bothered him so he could not sleep.

On December 21st, I had Dr. Hunt, the neurologist, to see him, on account of his persistent headaches. Dr. Hunt thought, owing to the fundus lesion and the occipital headaches, that the man had, in all probability, a brain tumor, possibly in the region of the pons, but did not advise any operation at that time.

The patient was not doing well, so I got Dr. Craig to open up his ethmoids and sphenoids again, and also got him at this time to drain the antrum of Highmore. His temperature was 100.6°

This operation was done on December 29th. December 30th, patient still complained of severe pain in the head. By January 4th, patient felt more comfortable, and on the night of January 4th slept fairly well. The treatment of applications and irrigations for his sinuses was kept up. Ever since December 30th the patient had shown marked improvement. January 5th the patient left the hospital, against my wishes, as he said he felt so much better. He continued to come to the clinic, however.

On January 14, 1918, I showed this patient at the meeting of the New York Ophthalmological Society. None of the members present questioned the diagnosis, even at that time, as the swelling and œdema were still very high. There was some difference of opinion as to the ætiology; some of the members thought brain tumor the cause, others thought it was due to a kidney lesion, while some of the members agreed with me.

I have contended all along that it was a case in which the clinical picture suggested the action of some severe toxin circulating in the blood. Four of my reasons for that are: first, headache; second, high blood-pressure in a man aged thirty-four; third, involvement of the kidneys; fourth,

marked œdema of the retina and papilla, with many hemorrhages. There was evidently some irritating substance in the fluid that filled the intervaginal space of the optic nerve. It is well known that after fracture, and more particularly after caries and necrosis of the body of sphenoid, infective meningitis may occur and lead to thrombosis of the cavernous sinus. Then why could not an empyema of sphenoids and ethmoids give us this toxin in the blood, which produced the kidney disease and the vascular disease of blood-vessels of the brain? And as the skull is unyielding, increased intracranial pressure arises as a result of the transudation of lymph and serous effusion, so that the lymph of the brain flows off to the spinal cord and also forward into the intervaginal space of the optic nerves. Owing to the increase of intracranial pressure, the neurologist advised against a lumbar puncture.

February 15th, the patient has shown gradual improvement, the hemorrhages are absorbing, and the swelling and œdema of the optic nerve and retina are not so great, V. O. D. 20/30; V. O. S. 20/30. Blood pressure 170. Patient comes to the clinic regularly and receives treatment also for his sinuses. March 16, 1918, I had a chemical analysis made of his blood at Post-Graduate Hospital. Urea 21.2; uric acid 7.4; creatinin 2.0; sugar 0.149. His urea and uric acid are still a little high, but his creatinin content is normal. Dr. Killian assures me that his uric acid and urea will be normal soon, and he considers the man out of danger. His kidneys are normal now, according to the test made of his urine in the pathological laboratory of the Infirmary.

The more recent quantitative micro-chemical studies of the blood have indicated that among the non-protein nitrogenous constituents the estimation of creatinin is likely to be of special service from a diagnostic and prognostic standpoint. In other words, the accumulation of creatinin in the blood has shown itself to be a useful index of renal insufficiency. The handicap of high creatinin accumulation, the kidney is, apparently, never able to overcome. Myers and Fine, who have accumulated extensive statistics on this subject, put the range of normal creatinin from 1 to 2.5*mg* per 100*cc* of blood.

March 18, 1918, I showed this patient at the Section on Ophthalmology of the New York Academy of Medicine. Again there was some difference of opinion as to the cause of the trouble.

Blood pressure now systolic 150, diastolic 100. The patient now has normal vision, normal fields for form, and slight contraction for colors, with enlargement of blind spot. The patient has no scotomata. The hemorrhages in the

retina and the œdema in the nerves and retinæ have been gradually absorbed. The surfaces of the disks appear untransparent and dense. The edges of the nerves are irregular and uneven, and the nerve heads look larger, showing a hyperplasia of connective tissue on the nerve heads and surrounding retinæ. Also there are some white streaks along the veins and arteries near the disks. The fundus of each eye, considering the severe inflammatory affection, presents a beautiful picture and one that is seldom seen.

April 1st, the patient has returned to work and feels fine. Blood pressure, systolic 140, diastolic 85. Now what else could have brought about such a remarkable recovery unless it was some severe toxin circulating in the blood, produced by the empyema of ethmoid and sphenoid sinuses? His kidneys had no treatment while in the hospital, except for two weeks before the operation, then I had him on a milk diet, rest in bed, and magnesium sulphate in the mornings. With that treatment his headaches increased and his vision gradually declined. The rapidity with which the eyes and kidneys cleared up, the writer thinks, leaves no doubt as to the cause of the trouble. It only goes to show the importance of making a thorough investigation when the ætiology is obscure.

Birch-Hirschfeld recently reported three cases of optic neuritis due to ethmoiditis. Good results were obtained by treatment directed to an empyema of the ethmoids. This case of mine is all the more interesting as at least 80% of double choked disks are due to tumors of the brain, either large or small, and is highly significant of intracranial disease.

One word in regard to differential diagnosis of papillœdema. In brain tumor cases: (1) The papilla is swollen out into a mushroom-like protrusion so as to appear enlarged but still well defined; (2) the choked disk is double-sided and not combined with retinitis. Whereas in cases due to focal infection we have:

I. More inflammation of the retina.
II. More œdema of the retina.
III. Generally there are many hemorrhages in the retina.
IV. We have a papillitis of the choked-disk variety.

THE INTIMATE RELATION BETWEEN THE EAR AND THE EYE AS SHOWN BY THE BÁRÁNY TESTS.

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IT was not until the last century that the knowledge of the relation between the ear and the eye became recognized. It is of interest to note that the very first work on the relation of the ear to nystagmus and to equilibrium was brought forth about the same time by two men who were working entirely independently. In 1825 Fleurens made excisions of portions of the labyrinths of animals and noted that it caused movement of the eyes and definite disturbances of equilibration. Perkinje at the same time conducted experiments in turning human beings and likewise observed the resulting nystagmus and vertigo. The efforts of all the earlier investigators were entirely along scientific lines; the practical application of these investigations fell to the lot of the Vienna school of otologists; in the past ten years, Robert Bárány especially has done his monumental work in extending the field of this study into the realm of neurology. Bárány received the Nobel prize of 1915 and he is to be regarded as the pioneer in the clinical application of this study of the ear and the central nervous system.

The purpose of this paper is to call to the attention of the ophthalmologist the governing power of the ear over eye-movements and equilibrium. It is not yet generally recognized that the ocular mechanism is dependent upon the ear stimuli for precision of movement. Steadiness of central fixation is made possible only by normally acting ears. Tonic impulses from the right ear continually tend to draw both eyes

to the left, and tonic impulses from the left ear continually tend to draw both eyes to the right. This is definitely proven in that a sudden loss of the function of the right ear invariably results in a rotation of the eyes to the right, because the tonic impulses tending to draw the eyes to the left are impaired, and there is a resulting spontaneous nystagmus to the left. This may be further demonstrated experimentally by the use of electricity. In applying the galvanic current to the right ear, by the use of the anode, which depresses its function, there results a drawing of both eyes to the right with a resulting nystagmus to the left. The kathode stimulating the right ear causes a drawing of both eyes to the left with a resulting nystagmus to the right. These diametrically opposite phenomena can be produced merely by reversal of the current. It is thus shown that ocular equilibrium in the same way as bodily equilibrium is dependent on normally functionating ears.

Aside from the production of nystagmus in this experimental way, the ear in many animals has a decided influence on ocular rotations. Barthels (*Klinisch Monatsblatt f. Augenheilkunde*, Sept., 1914) makes the statement that rabbits have no trace of voluntary fixation of the eyes, but that ocular movements depend entirely upon stimuli from the auditory apparatus, and that section of the acoustic nerves in rabbits produces complete loss of eye-movements. In extremely young children he says it is impossible to produce rotary nystagmus and, although the auditory apparatus is already exerting some influence, the results of ear-stimulation are irregular eye-movements. In those who have been totally blind for a long period of time, showing the peculiar searching movements which Barthels calls "nystagmus of the blind," these, he says, show much less reaction to aural stimulation than do normal individuals, and he believes that these purposeless movements, which have developed because the blind person is not aware of the position of his eye, act against the nystagmus induced by the aural stimulation, and he concludes from this that the nystagmus of the blind is entirely separate from that produced from the ear.

It is now definitely known that the ear consists of two organs of distinct and separate function—the cochlea which is the

organ of hearing and the vestibular labyrinth which is the sense-organ of balance. The equilibratory portion of the ear consists of two tiny sacs known as the utricle and saccule and of three semicircular canals; the utricle takes cognizance of movements in a linear direction antero-posteriorly, and the saccule of movement in a lateral direction; the semicircular canals are so constructed as to detect *rotary* movement of the body in all conceivable planes. The "horizontal" canal detects movement in a horizontal plane; the two vertical canals—the "superior" and "posterior"—always work together and detect movement in the sagittal, frontal, or intermediary vertical planes.

The new ear-tests consist of experimental stimulation of these semicircular canals. This may be done by revolving a person in a turning-chair, or by douching the ears with either cold or hot water, or by applying the galvanic current to the ears. Such ear-stimulation produces certain definite phenomena—a rhythmic nystagmus and a subjective sensation of turning—vertigo. The two and only two definite reactions from ear-stimulation are nystagmus and vertigo; because of this vertigo, the patient falls in a definite direction, and also when he attempts to find with his finger or his foot an object he has previously touched, he is unable to find it but "past-points" to the right or left, above or below, depending on direction of the vertigo. These various phenomena are invariably present in normal people, and furthermore always follow definite laws. These findings can be as definitely studied and recorded as can central vision or the field of vision. For example, turning the individual ten times to the right at a speed of two seconds to each turn, with the head in the upright position, stimulating both horizontal semicircular canals, produces a horizontal nystagmus to the left of twenty-eight seconds' duration, a subjective sensation of turning to the left, and "past-pointing" to the right. Douching the right ear with cold water 68°, head upright, stimulating the vertical canals, produces after forty seconds of douching a rotary nystagmus to the left, a sensation of falling to the left, "past-pointing" to the right, and falling to the right. Hot water produces exactly the opposite phenomena. Turning produces a mechanical movement of the endolymph

within the semicircular canal; douching the ears with cold water produces a chilling of the outer portion of the internal ear, causing a lowering of the specific gravity of the endolymph; it therefore drops and produces a circulation downward. Hot water causes the endolymph to flow in the exactly opposite direction—upward. Such is the complete control of the ear over the eye motion that a nystagmus of any type and in any direction may be produced "to order" by appropriate ear-stimulation. The eyes are always drawn in the direction and in the plane of the endolymph movement.

If stimulation of the semicircular canals causes the eyes to move, it is evident that there must be nerve-paths between the ear and the eye-muscles. Similarly a stimulation of the semicircular canal produces a conscious sensation which is called vertigo; it is evident that there must be a nerve-path between the ear and that portion of the cerebral cortex which receives this impulse. The recognition of the ear as the chief equilibratory organ is so recent that most of the intracranial pathways are still undetermined. A study of over 600 clinical cases, including a considerable number of operations and autopsies, in the department of Neuro-Otology at the University of Pennsylvania has brought out certain facts in regard to these pathways, some of them definite, others needing further analysis. Our present belief, in fact, is as follows:

1. The fibers from the horizontal semicircular canals pass through the VIII. nerve, enter the brain-stem at the junction of the medulla oblongata and pons, and continue directly to Deiters' nucleus and there divide into two pathways:

(*a*) The vestibulo-ocular tract concerned in the production of the nystagmus. These fibers go from Deiters' nucleus to the posterior longitudinal bundle, through which they pass to the various eye-muscle nuclei, from which, through the III. and VI. nerves they are distributed to the eye-muscles themselves.

(*b*) The vestibulo-cerebello-cerebral tracts responsible for the vertigo. From Deiters' nucleus this path enters the cerebellum through the inferior cerebellar peduncles to the three vestibular cerebellar nuclei of the same side, from which it proceeds upwards through the superior cerebellar peduncle and continues to the cerebral cortex from both sides, but more

particularly the opposite side, through the crura cerebri. The cortical areas which receive these fibers are postulated by Mills to be in the posterior portion of the second temporal convolutions adjacent to the cortical arens for hearing.

2. The fibers from the vertical semicircular canals have a very different course: after passing through the VIII. nerve they immediately ascend into the pons and at a point above the middle of the pons they have a division into two pathways similar to the division of the horizontal canal fibers at Deiters' nucleus.

(*a*) The vestibulo-ocular tract, the fibers entering the posterior longitudinal bundle to be distributed to the eye-muscle and finally to the eye-muscles themselves.

(*b*) The vestibulo-cerebellar-cerebral tract reaches the cerebellum through the ***middle*** cerebellar peduncle, entering the cerebellar nuclei of the same side; from this point the pathway is identical to that of the fibers from the horizontal canal, through the superior cerebellar peduncle to the cerebral cortex of both sides.

The internal ear and these intracranial pathways constitute our conception of the "vestibular apparatus," and a knowledge of this is of use to the ophthalmologist in the study of ocular palsy and spontaneous nystagmus.

From the standpoint of the clinician it is, of course, the neurological aspect of this recently acquired knowledge that is most valuable, and it is to the neurologist that the result of the tests of the condition of these tracts has been most useful. A greater stride has been made toward the accurate localization of cerebral lesions by the knowledge thus acquired than by any other one thing brought to the attention of medicine in many years.

The ophthalmologist is primarily concerned with the so-called "nystagmus tract," and has only a collateral interest in the pathways producing vertigo. Many cases of muscle paresis and paralysis have much of interest added to them by a study of the ocular motions produced by ear-stimulation, and at times information of great clinical value is obtained: for example, given a patient with loss of conjugate deviation to the left, should stimulation of the right ear draw both eyes to the left, it is proven that the tracts from the nuclei to

the end-organs are unbroken and the lesion is supranuclear.

No case of nystagmus can now be considered as completely studied unless the results of ear-stimulation have been noted, and this frequently throws much light on the subject, both as to the site of the irritation producing the nystagmus and as to the prognosis. Illustrative of this may be cited two cases seen by us not long ago in the Children's Hospital; both were brought in unconscious with rather vague histories, both apparently were somewhat similarly affected, each running a high temperature with constant purposeless nystagmic motions of the eyes. They were believed to be the results of intestinal intoxication. An eye-examination elicited nothing, the ocular structures being normal. Ear-stimulation with cold water in one case produced a normal response, the purposeless motion ceasing immediately and a rhythmic nystagmus taking its place; in the other case no change took place in the character of the ocular rotations after ear-stimulation. In the first case, an opinion was expressed that the child's cerebral structure had not seriously suffered, and from this it seemed that recovery was possible; in the second case, since the ear-stimulation did not pass through the pathways in sufficient strength to cause a normal response, it seemed that there had been much damage in the brain tissue, and that the child might not recover. Each prognosis was proved by the outcome to be correct.

In cases of muscle paresis it has occurred to us that ear-stimulation by means of electricity might be used therapeutically, as the alternate pull and relaxation of the changing current would possibly succeed in stimulating to action what nerve-fibers there were remaining intact and shorten the time of recovery. We have had no opportunity of trying this, so it is in the realm of theory, but we intend to work along this line in the near future.

SOME PHASES OF THE VESTIBULAR NERVE PROBLEM.

(1) DEVELOPMENT OF THE VESTIBULAR TRINITY. (2) FUNCTIONS OF THE CEREBELLUM. (3) THE PENDULUM MOVEMENTS OF THE EYEBALLS.

By JOHN DUNN, M.D., Richmond, Va.

THE author wishes to say that without study of Bárány's splendid contributions to the subject of vestibular nystagmus, the suggestions made in the following paper would have been impossible for him.

Sitting in a Bárány chair, with my fists balled on my knees and my head bowed forward until it rested on them I was turned five times to the right. (One of the well-known tests used in the examination of candidates for aviation.) In my efforts to sit up I felt a slight pull to the right at my right ankle joint. This pull was progressively greater at the knee, the hip and shoulder, and by the time my effort was at its height, I felt as though I were made of rubber and my body and head were bent violently to the right. This increasingly great pull from my ankle joint upward to my head was so distinct that it resulted in my asking myself the question, "Why should this be so?" Standing upright I now bent my head rather violently towards the right shoulder, and I found that I was reproducing exactly the body and head and extremity movements which I had experienced in my effort to sit up in the Bárány chair, only in reverse order. As in the former experiment, so in the latter, I could feel the efforts of fixation at the ankle, knee, and hip, while my body and head were making a still greater excursion.

Several thoughts at once suggested themselves. Why should the head fall through a greater arc than the shoulder, the shoulder than the body, the body than the hip, the hip than the knee, the knee than the ankle? Why was the whole body, including legs and arms, not drawn an equal distance to the right? Why to the right and not to the left? Why should ocular nystagmus form any part of this curious falling movement? Why should there be bound up together the vestibular triad, the body and extremity movements, the nystagmus or ocular movements, and the disturbed flow of the endolymph over the cristæ? To say that the latter does evoke the other two is no answer at all. It is merely stating a fact. Why should it do it? Why is it not equally possible to evoke from the eye movements of the endolymph and the body and extremities? Or from these latter the other two? These are fair questions, and upon their proper explanation is founded the understanding of the vestibular syndrome, so far as it contains only the above three elements.

"It would be important if the influence of head movements upon nystagmus should be exactly studied" (Bárány, *A. of O. R. and L.*, vol. xxi., No. 1, pp. 107–8).

When I stand erect and bend my head toward the right shoulder, what takes place every time I do so? What has taken place every time I have done so since earliest infancy? In the first place I bring into play the will. In the second place there is a contraction of the dextro-turning cervical muscles. In the third place there is a relaxation of the sinistro-turning cervical muscles. In the next place, in order that *the head may be accurately turned in exactly the willed degree*, there takes place a yielding to the right of not only the head, but of the body, of the shoulder and arm, and of the hip with the knee and ankle. To prove this beyond question all that is necessary is to stand erect and turn the head rather violently toward the right shoulder, fixing at the same time the attention on the changes in position which the body, shoulder, etc., make. The sensations at the various joints tell of their participation in the head movements. We experience at the same time a counter-balancing relaxation of the left side of the body, etc This, however, is not all that takes place. Both eyes rotate (not turn) in the opposite direction, *i. e.*, to the left, and there

is a disturbance of the endolymph of the vertical semicircular canals. From earliest infancy through life these three things take place with varying degree of intensity every time the head is bent towards the right shoulder, and in reverse directions whenever the head is bent towards the left shoulder. There has thus come into existence the vestibular trinity, and thus too have come into existence those intimate and excessively accurate nervous connections which are necessary for the regulation and coördination of these three elements of the vestibular syndrome. The understanding of these simple facts make clear in a wonderful way the vestibular problem. We can see at once the necessity for body falling, for past-pointing, for directional nystagmus. We can see the necessity for rotary nystagmus and for horizontal nystagmus. We can see the necessity for special branches of the vestibular nerve to the vertical canals and of the special branch to the horizontal canals. If we hold our head in the so-called optimum erect position and turn it to the right, what takes place? The eyes move in the opposite direction, *i. e.*, to the left. There is an excess of movement in the endolymph, confined to the horizontal canals. There are necessary body and extremity movements and adjustment, similar in kind, although less in degree, to those aroused by the turning of the head toward the right shoulder. Turn the head to the left and a similar trinity is brought into existence but in the opposite direction. The individual contributions to this vestibular trinity come into existence all through life each time the head is revolved about the vertical axis. And so again come into existence the intimate nervous connections necessary for their coördinated continuance. It so happens that anatomically the peripheral nervous contributions at work exist in a small compass only for the vestibular nerves, hence it is possible by peripheral excitation to reproduce the other two elements of the trinity only in the case of the vestibular nerve. In considering the body and extremity movements, the ocular movements, and the directional disturbances of the endolymph, we must bear in mind that they vary greatly in the ever-changing calls for varying the position of the head. The three primary combinations, around which all the others center, are those which result from movement of the head about its three main axes.

How came it about that there is necessary for the proper correlation of these three vestibular elements so elaborately developed an organ as the cerebellum? Bárány (*loc. cit.*) says: "the chief task of the cerebellum is to tone up and increase the efficiency of the musculature." Exactly what these words are meant to convey I do not know. They are certainly too inexact for me to get from them any clear idea of the functions of the cerebellum. This organ has functions as definite as those of the cerebrum, and until we know accurately what these are we must continue to talk loosely of many features of the vestibular syndrome. As I see it, the cerebellum so regulates the muscular control of the spinal column and of the various joints of the extremities as to make possible the exact performance of any desired, *i. e.*, willed, movement or movements. For instance, with my right arm extended I will to pronate it. That this may be accurately done in the right direction and with the rapidity I may will a certain exact fixation of the neck, of the shoulder joint, of the elbow joint, of the wrist and finger joints has to be made, otherwise the willed movement of pronation is inaccurately done, over- or under-done. A proper understanding of this fact makes easy the interpretation of the various disturbances of the cerebellar function; hypermetria, asynergia, adiadokokinesia, tremor, disturbances of writing, atony, hypertonia, hemiparesis, past-pointing, etc. That there should be definite cerebellar centers with the functions ascribed to them by Bárány and in the positions claimed by him is entirely in accord with this view of the purpose of the cerebellum. There is no evidence that the cerebellum originates movements. Its centers respond to sensations reaching it from without. The nervous current, so to speak, has to be turned into its centers to arouse its responding activities. From what source or sources may these responding activities of the cerebellum be put into action? I will to raise my right arm. In this instance the source of the impulse is in the left cerebral cortex. I liberate impulses in the right arm cerebral cortical center. These impulses pass through the internal capsule. A part of them pass unbroken through the crus and the crusta of the pons into the anterior column of the opposite side of the cord and so on. Another part passes to the pontine nuclei. Why

to the pontine nuclei? And whence from there? These impulses on reaching the pontine nuclei are sent to the cerebellum. For what purpose? To adjust the spinal column, shoulder, and neck so that the arm can with exact precision be raised. This is as clearly as I am able to state the two elements of all-willed impulses from the cerebral cortex. One element, the direct, is to accomplish the definite willed movement or movements; the other, the indirect, enters the coordinating nuclei of the pontine region and are transmitted to the cerebellum where they originate impulses whose result is to adjust the body stem and the joints so that the willed movement can accurately take place; these impulses result in the primary cerebellar response, *i. e.*, the willed response of the cerebellum. There is, however, another source from which cerebellar activities can be aroused, and this represents the purely reflex side of the cerebellum. This source is all the sensory nerves which take notice of voluntary muscular activities, *i. e.*, the sensory nerve supply to the muscle sheaths, to the tendons, to the ligaments about the joints, to the fascia and to the skin. The prime object of these peripheral born cerebellar excitants is to maintain the result of the cerebral willed impulse until it is released and another cerebral born impulse takes its place. *This is the more important raison d'être of the cerebellum.* For instance, I will to take a book in my hand, I will to sit in an easy chair, to cross my legs, to get my body in a comfortable position, to hold the book at an agreeable distance from my eyes, etc. These acts having been accomplished, I read the desired length of time, interfering with the fixity of my comfortable position only to will to turn the pages of my book. The maintaining of my reading position is left entirely to the peripheral born cerebellar excitants which without further exercise of cerebral activities maintain reflexly and subconsciously the position of my body and extremities. It is out of place here to elaborate the details of this view of the functions of the cerebellum. It is, however, necessary to have it here clearly in mind. The cerebellum can originate no muscular movements in the sense in which the cerebrum can. The cerebellum has no tonic influence over the muscular system. The muscular atony, etc., which result from deprivation of cerebellar function

are purely manifestations of disuse of the parts concerned. The cerebellar functions just mentioned, *i. e.*, the primary or willed, and the guardian function, have charge of all of the voluntary muscular activities which originate in the exercise of the will.

Development of the vestibular trinity imposed upon the cerebellum a further intricacy of detail that exact correlation of the movements of the head, of the ocular muscles, and of the endolymph of the semicircular canals might exist. Under normal conditions the willed turning of the head about the vertical axis arouses those primary cerebellar impulses, whose object is to adjust the spinal column, shoulder joint, etc., that the turning of the head may accurately be done; it arouses the ocular extended vagus action so that both eyeballs turn evenly toward the opposite direction, and lastly it arouses motion in the endolymph of the horizontal canals and this to a greater degree in one than in the opposite canal. (This latter owing to the shape of the two extremities of the canals.) Of these three elements none are directly under the will. The adjustment of the body and extremities, the ocular movements, and the aroused vestibular sensations occur independently of, and are beyond, the control of the will, when once the machinery for the contraction of the neck muscles has been set in action; all of them are unwilled results of the willed movement of the head. That the unwilled coördination of these three elements may take place there have been developed connections between the cristæ of the horizontal canals and both the cerebellar cortical centers and the vagic ocular centers. When the head is turned toward either shoulder similar conditions are aroused except that in this case the movements of the eyes are rotary and that the endolymph of the vertical canals is brought into play. And similar nervous connections are made between the cristæ of the vertical canals and the cerebellum and the vagic ocular nuclei. Inasmuch as when the head is turned to either shoulder the trunk and extremities require a more elaborate adjustment of the muscles than they do when the head is turned from side to side, the central connections of the vertical cristæ are more elaborate than are those of the horizontal canals. This is proven by the wider head, body, and extremity movements

when the endolymph of the vertical canals are artificially stimulated than those that occur when the same degree of stimulation is applied to the endolymph of the horizontal canals. There is still a third axis about which the head can be moved. The head can be moved forwards and backwards. Pretty much all comment upon this fact has been omitted in the discussion of vestibular nystagmus. It is none the less of the first importance if we are to have a proper conception of the subject. When the head is moved backwards and forwards in the antero-posterior plane it is accomplished with far less call upon the trunk and extremity musculature than when it is turned in either of the other two planes. What is, however, of more importance is the fact that the muscular response is equal for the two sides. This is entirely different when the head is either turned laterally or toward either shoulder. Here, as we have stated above, the contraction of one side is accompanied by a relaxation of the other, and this relaxation is under the control of the cerebellar centers. Moreover, when the head is moved up and down, whatever disturbances of the endolymph take place they are equal for the two sides. Therefore, moving the head in the antero-posterior plane can result in neither past-pointing, nor in the production of nystagmus. Moreover, when the head is dropped forward both eyes move directly upwards, and when it is thrown backwards both eyes move directly downwards. The will cannot move both eyes at the same time directly upwards or downwards. This can be accomplished only by suppression of the willed control of the ocular muscles and by turning them over to the vagic control. (It is both of interest and importance to note in this connection that vertigo can be produced by moving the head directly upwards or downwards. This fact will be commented upon later.) In reading Bárány's paper (*loc. cit.*), I rightly or wrongly came to the conclusion that he teaches that the cerebellar cortical centers which he so accurately describes and locates all directly respond to vestibular peripheral irritation. My own idea is entirely different from this. I believe that only those cerebellar centers directly respond to vestibular peripheral irritation which have been developed along with and are of necessity a part of the vestibular trinity complex, *i. e.*, those centers whose hyper-excita-

tion produce body falling and past-pointing of the type made manifest by irritation of the cristæ. The cerebellar centers for pronation, supination, flexion, and extension, etc., are developed in connection with the primary and guardian cerebellar functions, as described above, and are directly associated with the cerebral motor cortical centers. This is a very important distinction and without its full appreciation no correct understanding of cerebellar function is possible. That tumors, etc., of the cerebellum disturb the functions of both the vestibular and primary cerebellar centers is easily appreciated when we bear in mind that their size is such that they may disturb both sets of fibers or centers.

What has been written above of the vestibular syndrome in relation to its genesis from movements of the head is further borne out by the fact that the facial muscles, the tongue, and the voluntary laryngeal muscles do not participate in it. In turning the head in its various directions, none of these muscles are brought into play, and so no inter-communicating cerebellar centers for their associate adjustment have been developed. There is another fact which has not received proper recognition in descriptions of the vestibular syndrome. This is the tendency of the head to over-fall in the direction of the slow movement of the nystagmus.

Let us consider the eye movements. An explanation of past-pointing and body falling has been given. An explanation of the details of the vestibular nystagmus is more complex. In the former case, we are dealing with a disturbance of movements in the domain of those parts of the brain in which are exercised the powers of the will: the cerebral hemispheres and cerebellar hemispheres for the extremity movements, the cerebral hemispheres and the vermis for the body movements. Of these two, the former belong to a later period in the development of the brain than the latter and this accounts for the position of the centers for movements of the trunk muscles. Around the vagi nuclei the whole of the brain in its future elaborate development arose. In man, the centers for the ocular movements are pushed, as it were, relatively far forward along the brain stem. This tells of two things: first, that the eye centers, as developed in the human family, are the latest addition to the brain stem; second, that room had

to be made for the connections with the fibers which regulate the willed movements of the eyes. The primal eye was a part of the vegetative system and responded to impulses from the vagus centers. In its elaboration by development it has remained both for its internal mechanism and its external muscular apparatus under control of the vagus system, although by its later connection with the cerebral cortex, it has yielded a share in the control of its rotation movements to the domain of the will. Thus it has come about that excitation of the vestibular terminals sends impulses to both the vegetative ocular nuclei and to those parts of these nuclei which have later developed as the result of the addition of a willed control over the external ocular apparatus. Thus again it has come about that the nystagmus has a double element. The one which, as in the case of nausea and vomiting, results from over-excitation of the primary stem centers and becomes manifest whenever this hyper-excitation takes place, provided the stem centers are still functionating. This explains "the deviation of the eyeballs in the direction of the slow movement even in deep unconsciousness," for here the willed control of the eye muscles has been removed, while the vagic control is still active. The second element of the nystagmus results from over-excitation of the centers which have developed as the result of willed control over the external ocular muscles. These are similar to the adjunct cerebellar centers for the willed movements of the body and extremities. These centers must be in the cerebellar cortex and are probably in the paleocerebellum. The manifestation of their hyper-excitation requires, as in the case of past-pointing and body falling, a call from the cerebral cortical centers. Their manifestation follows the same rules which determine the direction in past-pointing and body falling. To understand the double movement of the nystagmus, the slow and rapid, it is necessary to know what takes place with the eyes when the head is turned from side to side, up or down, or bent towards the right or left shoulder. Little has found its way into the textbooks about the pendulum movements of the eyes. These may be best demonstrated as follows: Hold a mirror before the face and while looking into the mirror turn the head from side to side. It will be seen that the eyes move in a direction opposite

and with a rapidity equal to the movement of the head. They maintain their parallelism, but their movements from side to side have an evenness, a lack of jerkiness, which is altogether absent when the head is held fixed and the eyes are turned from side to side. Their movement resembles that of a pendulum in its evenness. In this experiment the willed control of the eye muscles is entirely suppressed. There is no consciousness of this ocular movement. On the other hand, when the head is fixed and the eyes are turned from side to side, not only is there consciousness of the movement of the eyes, but there is a visibly demonstrable jerkiness of them. In the latter experiment we see the efforts of the eyes to respond to the will. The former gives the most complete demonstration we have of the extended vagus control over the external ocular muscles. The evenness of the pendulum ocular motions is something that cannot be obtained by the will when the excursions of the eyes are either extensive or rapid. Witness the tendency to nystagmoid movements when the eyes are turned by the will, *e. g.*, either to the extreme right or left; and, as just mentioned, the accompanying jerkiness of their movements, which are so great as to arouse within the brain consciousness of them. The pendulum movements belong to the period in the development of the eyes when they turned automatically to the light as such without references to interpretation of form. It is further to be noted that the pendulum movements, in whatever direction they occur and whether up or down, from side to side, or rotary, are opposite the movement of the head; while, on the other hand, the fixing movements which occur at the end of the pendulum movement is in the direction of the head movement. The former is an expression of extended vagus action; the latter of cerebral action. Both are brought into play with every movement of the head. Applying to the ocular movements the facts stated about the past-pointing of the extremities and body falling, we can see that the direction of the nystagmus is determined by the change in the position of the head and only secondarily by the change in directional flow of the endolymph in the semicircular canals. The slow movement of the nystagmus, to repeat, represents the response to irritation of the vagi nuclei and correspond to the pendulum motion.

The rapid movement of the nystagmus represents a past-pointing of the eyes similar to the past-pointing of an extremity and necessitates an element of will for its accomplishment. Or to express it another way, in turning the head rapidly from side to side there is suppression of the willed control of the external ocular muscles; while with the head fixed if we turn the eyes rapidly from side to side there is suppression of the vagic control.

It may not be out of place here to discuss one or two of the details of the vestibular trinity. When artificial disturbance of the endolymph of, say, the right horizontal semicircular canal has been induced, there appears, even though the head be kept from movement of any kind, a horizontal nystagmus. At first thought this fact would seem to overthrow all that has been said about the development of the vestibular trinity. There is no motion of the head and yet disturbance of the guardian impulses, *i. e.*, those originating in the cristæ are able to bring into play ocular nystagmus. Apparently all three elements of the trinity should be always brought into play when one of the others is. Why should this disturbance of the vestibular terminals to the crista in the right horizontal semicircular canal not also induce, even when the head is kept motionless, movements of the body or extremities, as well as ocular nystagmus? In the first place, the impulses from the excited crista find their way to the vagic ocular centers, which as we have tried to show are in no way under the control of the will and are disturbed by peripheral born impulses in just the same manner as are the other vagic nuclei whose over-excitation results in nausea or vomiting. Thus it comes about that the eyes are turned to one side or the other, dependent upon the nature of the excitant applied to the endolymph of the crista. This vagic turning of the eyes takes place, whether the eyes are open or shut. When the eyes are open the will makes an effort to overcome this vagic movement and nystagmus is the result. If the will turns the eyes in the direction of the vagic movement the nystagmic movements either cease or are markedly less apparent. As a further result of stimulation of the vestibular terminals to the crista of the right semicircular canal impulses are sent to the cerebellar centers, central and cortical. Of these latter, only

those receive an over-stimulation which have been developed as a part of the vestibular trinity, *i. e.*, those which are concerned in those regulations of the body stem and joints of the extremities which make accurately possible the willed movement of the head through the action of the neck muscles. Although this is briefly discussed elsewhere in this paper, it is here repeated because of its importance. I have shown that the ocular nuclei are so constructed that, although the microscope cannot demonstrate the details, it is possible to excite the vagic elements without exciting the cerebral elements. In the same way in the cerebellar cortical centers, as well as in the central nuclei, it is possible to bring into play the elements which have been developed as a part of the vestibular syndrome without exciting those parts which have been developed to make accurately possible the demand of the will, *i. e.*, the responses of the varying parts of the musculature to impulses from the various cortical centers.

Movements of the eyeballs occur without causing the sensation that external objects have at the same time a corresponding motion. This is true whether we consider the normal movements of the eyes or the uncontrollable nystagmic movements which occur from certain causes, *e. g.*, albinotic nystagmus and optic neuritic nystagmus. With vestibular nystagmus external objects have a movement.

The next question that comes up is, Can there exist, while the eyes are at rest, a constant subjective sensation that all objects in the field of vision are in motion? The two following cases are of interest.

Mrs. L., aged 38. Six years before she came under my care she suddenly had an attack of vertigo, so severe as to cause her to fall. Since that time she has never been free from vertigo, the intensity of which has varied greatly. At the time of her first attack she did not lose consciousness. In subsequent attacks she has done so "for a moment or two." In the ten days prior to her first visit to me there had been three of these major attacks. This is more frequent than is usual. The attacks are followed by the sensation of a chill. After the Bárány tests in which she fell and past-pointed normally, and in which the nystagmus appeared normal in direction, she was ill for three days, feeling all the time "the boat motion." It was two hours before

she could stand erect. Both ears respond equally in about fifteen seconds to a cold caloric test and the sensation of dizziness and vertigo, produced by this test, was intense, so great indeed that the patient cried piteously. She was much nauseated. There was no visible nystagmus produced by this test and no demonstrable past-pointing. It was *as though the cold produced its vertigo and nausea effect before the semicircular canals became affected.* She is always conscious of the right ear; as though there were "something deep down in it." Hearing, both ears, normal for all tests. Eustachian tubes patent. Drum membranes show nothing definitely pathological. When sitting erect, if she bends her head forward, ever so little, and looks forward and downward, the sensation aroused is as though the chair in which she is sitting and she herself are moving backward. When she lies down everything begins to whirl, she thinks worse when she lies on her right side. Eyes: vision normal. Fundi and media normal. Pupillary reactions normal. No exo-, eso-, or hyperphoria. No areflexa corneæ. She feels as if she wants to close the right eye; as though the lids want to drop. No visible loss of function of upper lid. No nystagmus, even for forced movements of eyes to right or left, up or down. No unsteadiness of eyeballs can be made out, either when a light spot in the cornea is watched under a magnifying lens, or when the optic disk is under observation by the direct method. Movements of the head to right or left, do not increase dizziness. She, however, is distinctly conscious all the time of her right eye. She feels as though she were "going blind in it." Color sense normal. Fields normal. The most interesting symptom, however, for us here is that everything in front of her is in motion. For instance, a pencil held vertically at the distance of three feet in front of her has a blurred outline and is never still. The pencil "moves from side to side like the pendulum of a clock," within, however, very narrow limits. This blurring of the outlines of all objects looked at causes a sense of indistinctness in everything, and makes her feel as though "she were going blind." General examination: Patient has worn for a considerable time a supporter for gastroptosis. Has some enlargement of the thyroid; probably mild form of hyperthyroidism. Pulse 90. Patient was operated on several years ago for appendicitis, since which time her right inguinal region has never been entirely free from pain and she complains of many attacks of distention. Family physician says she has slightly retroverted uterus and some stenosis of cervix uteri. History of long-standing constipation.

Without going into the further history of the case there stand out several phases of it which interest us here. In the first place, the "neurasthenic" element is plainly visible, as demonstrated by the fact that the cold caloric test is equally over-active for both the right and left ears. In the second place, this test causes dizziness and nausea before any demonstrable effect is produced upon the fluid in the semicircular canals. Again turning the head from side to side does not increase the patient's symptoms, while bending it forward or backward increases them tremendously, so much so that the patient asked to be allowed to have her throat sprayed, instead of being required to gargle it, as the latter required her to throw her head backwards, while the former could be done while the head is in the upright position. The excited terminals are not those of the cristæ of the semicircular canals, for not all symptoms of their excitation are evoked by the various minor tests which so readily provoke dizziness and nausea. The absence of any deafness or noises in the ears, even in the ear of which the patient complains of a sense of fullness, shows that the cochlear branch is not involved. We are then forced to attribute the disturbance to our excitation of the third branches of the vestibular nerve, *i. e.*, the otolithic branches. This opens up the question whether the nausea we meet with in the various tests of function of the semicircular canals is to be attributed to over excitation of the cristal terminals or is caused by simultaneous involvement of the otolithic vestibular terminals. It seems not unlikely that the latter will prove to be the correct answer, and the same for the symptom vertigo. What is the cause of the "pendulum movement" of all objects looked at? Is it reflex excitation of the purely vagic element of the ocular nuclei? It further would indicate that the otolithic branches of the vestibular nerve belong to an earlier stage in the development of the nervous system than do the semicircular canal branches. It is a question further whether they have any direct connection with the cerebellar centers.

Mr. B., aged 42. Three weeks prior to examination had "a dizzy spell" with vomiting. Since that time the sensation of vertigo and unsteadiness of his body has, while gradually lessening in intensity, never left him. There

has been no return of the nausea. General examination reveals nothing of interest *save a patch of acute arteritis in the left optic disk.* Hearing: right ear, low and middle tuning fork not heard by air conduction. High fork heard well by air. Bone conduction normal for low and middle forks. Ringing in ear. Drum shows old deficiency in upper posterior quadrant. No discharge. Left ear normal. Eustachian tubes patent. Response to cold caloric normal. Patient is still "light-headed." If he turns his head suddenly, with eyes either open or shut, to one side or up and down or towards shoulders, vertigo immediately appears. Turning the eyes to extreme right or left produces a marked unsteadiness of the eyeballs, and in this movement there is visible a considerable amount of rotary nystagmus, as far as I could judge equal for a right or left turning of the eyes. Vision is normal. Pupils and media and external muscular balance normal. Everything in the visual field is "trembling like a mass of shaken jelly." There is no definite direction to the movement. This movement of all objects looked at produces a "sensation as though he was going blind." Attempts to fix small objects do not increase the movements of the objects looked at. Here we have a further demonstration of the fact commented upon, *viz.:* Of the nervous association of the lateral and up or down movements of the eyes with similar movements of the head. The absence of participation of the convergence movements of the eyes is a still further demonstration of the exactness of the nervous connection and control in the production of the vestibular syndrome. The more complete our knowledge of the elements of the vestibular syndrome becomes, the more clearly is it seen that each element is the result of the excitation of a definite set of nerve terminals, whose pathways and control connections are accurately made for the accomplishment of their several definite purposes. Only those ocular movements take part in the vestibular syndrome which are always, as shown above, brought into play with changes in the position of the head. The convergence movements and the iritic and ciliary movements, not necessarily participating in the movements of the head, do not appear as a part of the vestibular syndrome. This is repeated here as its full appreciation throws much light on the relation of the ocular movements and to disturbances of semicircular canal elements of the vestibular nerve. If a patient be seated in a dimly lighted room and a cold caloric test is done, in a certain proportion of cases the pupils will be seen to slightly contract. This pupillary response is not a part of the vestibular syndrome, but one of the manifestations of the pupillary skin reflexes. Again

in the case of Mr. B., when the eyes are directed forward, there is no visible demonstrable motion of the eyeballs. The eyes were carefully examined under a magnifying glass in several different ways. And yet everything seems to be in motion. The nature of this motion and of that in the case of Mrs. L., involved as they are in questions, which arise concerning vertigo are beyond the intended scope of this paper. Lastly Mr. B., although a phase of vertigo is constantly present when his eyes are open, has no sensation of nausea, not even when on rapidly turning his head the tendency to fall to one side appears. Compare in this connection the case of Mr. H., 65 years of age. Ever since he could remember the rapid throwing of his head backwards would produce vertigo—without inducing either nausea, vomiting, or the other so-called vestibular symptoms.

These two cases ask many questions. Foremost among them are, What are the functions of the utricle and of the saccule? Why has nature thought it best to subdivide the vestibular nerve so as to send some of its branches to the utricle and the vertical canals, and others to the saccule and the horizontal canal? In the order of development which has the priority, the vertical or the horizontal canals? Why should nausea be a sequence from disturbance of the ear? Why vertigo? (The latter seems to be easier to answer.) Do there pass centralwards from the cristæ, fibers some of which are destined to arouse stimuli in the oculomotor centers, others to assist through the cerebellar centers in stabilizing the head, spinal cords, and joints of the extremities for the fullest performance of willed movements of these parts? (If so, and the functional "tests of the canals" seem to indicate it then the cristæ are organs more delicately elaborate than has yet been understood.) Can nausea be induced by disturbance of the cristal nerve end alone? Or is nausea a manifestation of disturbance of the otolithic branches of the saccule? (And utricle?) What are the effects upon the endolymph beyond the semicircular canals of disturbances of the endolymph within them? These and many others equally important unanswered questions show the need for a clearer understanding of the mechanical problems of he endolymph and of the anatomical structures of the labyrinth. Until the tide of the endolymph is fully understood the literature of the vestibular nerve will continue to be filled with guesses.

THE LIGHT PUPILLARY REFLEX, ITS PATH, AND ITS ABOLITION CALLED IMMOBILITY OF THE PUPIL TO THE LIGHT REFLEX, AND REPORT OF A CASE OF UNILATERAL ARGYLL-ROBERTSON PUPIL, IN WHICH CONSENSUAL REACTION EXISTED IN BOTH EYES.

By Dr. ANTON LUTZ, Havana.

(*Concluded.*)

(*With Figure 9 in the text, and Text-Plate XV.*)

3. Efferent path for pupillo-dilatation.

The fibers of the ganglion cells of the centrum ciliospinale leave the cord by the ventral roots of its corresponding nerves, enter the rami comunicantes, and run through the ganglion stellatum and the ganglion cervicale inferius to the ganglion cervicale superius. Cl. Bernard (1858) showed that after the passage through the ganglion cervicale superius the troncus sympathicus cervicalis divides into two branches; one branch includes the vasomotor fibers which have no influence on the pupil, and which enter the plexus carotideus; the other branch includes the fibers for pupillo-dilatation, and goes through the cervico-Gasserian strand, independently of the plexus carotideus, to the ganglion Gasserii and passes thence into the first division of the trigeminus (nervus ophthalmicus), following the nasal branch which it leaves finally to enter the long ciliary nerves, thus avoiding the ciliary ganglion. The long ciliary nerves enter the eye on each side of the optical nerve, and running forward between the choroid and the sclerotic, pass through the ciliary body to be distributed to the iris. The experiment of Budge shows that after having cut the ophthalmic

branch (not the whole trigeminus), we can no longer produce dilatation of the pupil through excitation of the cervical sympathetic, and that the dilatation fibers therefore pass through the ganglion Gasserii in the nervus ophthalmicus. Braunstein (1894) extirpated the ciliary ganglion and produced mydriasis through excitation of the cervical sympathetic or of the nervuli ciliares longi. We must therefore conclude that the radix sympathica ganglii ciliaris consists in the vaso-contractor branch of the plexus carotideus. Angelucci Lodato found, after extirpation of the ganglion cervicale superius, degenerations in some of the large ganglion cells of the ciliary ganglion, as well as in ganglion cells scattered through the orbit. I think these ganglion cells must therefore be considered as intermediate stations of the vasomotor sympathetic. The experiments of Langley and Anderson (1892) with local applications of nicotine to the different sympathetic ganglia and injection in the blood show that the dilatation fibers run through the ganglion stellatum and ganglion cervicale inferius, and enter only in connection with the ganglion cells of the ganglion cervicale superius. Schultz (1898) showed that after extirpation of the ganglion cervicale superius, it was yet possible to excite the musculus dilatator iridis and that it needed some days before the degeneration of the post ganglion fibers reached the musculus dilatator pupillæ.

The terminal organ of the pupillo-dilatation path is the musculus dilatator iridis, the existence of which the physiological experiments of Langley and Anderson made probable in 1892. They introduced electrodes through a wound in the limbus corneæ, and produced electrically a radial contraction of the iris. Later (1898) Karl Grunert proved the real existence of the musculus dilatator iridis through splendid anatomical investigations.

Considering these facts we have the following

Chain of Neurons in the Pupillo-Dilatation Path.

1st. Unknown path of the different sympathetic fibers of the cranial nerves (fasciculus longitudinalis post.) through the mesencephalon and bulbus medullæ.

2d. Ganglion cells of the centrum ciliospinale, whose fibers

SCHEME OF THE PUPILLARY LIGHT REFLEX-PATHS

The scheme is incomplete in so far as it only shows the basal reflex-path. In order to avoid a too complicated design there are omitted: (1) the vasomotor reflex arc, which goes through the ciliary ganglion; (2) the cortical reflex arc, which begins in the corpus geniculatum externum, goes up and back through the radiation of Gratiolet to the calcarine fissure, where it finds its reflex arc in the stripe of Genari, and returning the same way descends to the pulvinar. The last step of it goes probably through the commissura cerebri posterior and is drawn in the scheme. Its existence is physiologically proven in so far as an electric excitation of the calcarine cortex produces marked miosis.

The basal reflex-paths consist of:

A. SENSORY AFFERENT PATH.
- (1) Cones and rods.
- (2) Multistratified bipolar cells.
- (3) Multistratified ganglion cells.

N. opticus, chiasm, tractus opticus, brachium conjunctivum.

Affected in:
- (a) Amaurotic light immobility.
- (b) No suppression of the consensual reaction.
- (c) Hemiakinesis with hemianopsia.
- (d) Hemiakinesis without hemianopsia.

B. PUPILLO-MOTOR CENTER.
(Deeper layers of the corp. quadrig. ant.)

C. EFFERENT PUPILLO-CONTRACTION PATH.
- (4) Fasciculus tecto-bulbaris (origin in corp. quadr. ant.).
- (5) Fibers of the oculomotorius (nucleus photomotoricus).
- (6) Nervus ciliaris brevis (origin in ganglion ciliare).
- (7) Muscle sphincter iridis.

Affected in:
- (e) Pure direct unilateral Argyll (with consensual reaction in both eyes).
- (f) Total unilateral Argyll.
- (g) Iridoplegia with medium mydriasis.
- (h) Iridoplegia with marked mydriasis.

D. PUPILLO-FIXATION PATH.

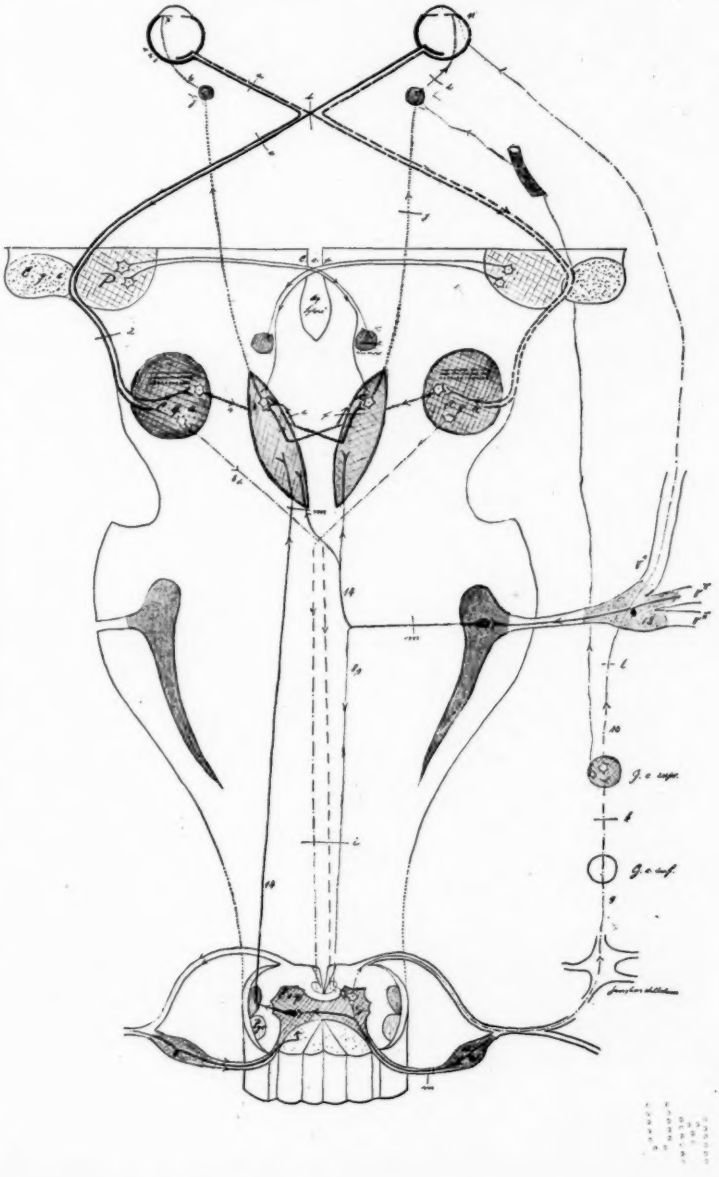
I. Active efferent path:
- (8) Fasciculus tecto-spinalis (origin in corp. quadr. ant.).
  Descendent fibers of the fasciculus longitudinalis post (origin in the sensitive cranial nerves).
  Spinal reflex arc of the sensitive branchial nerves.
- (9) Nervus sympathic. cervicalis (origin in centrum ciliospinale).
- (10) Nerv. sympathic. cranio-basalis (origin in ganglion cervicale supremum).
- (11) Musc. dilatator iridis.

II. Passive afferent inhibition path:
- (12) Sensitive end organs in the skin.
- (13) Ganglia spinalia, Gasserii and their homologues.
- (14) Ascendent fibers of the fasciculus longit. posterior.
  " " " " " latero-dorsalis Mahaimii.

Affected in:
- (i) Bulbar miosis.
- (k) Spinal miosis.
- (l) Hommer's miosis.
- (m) Abolition of the pupillary dilatation through pain sensation.

Also the basal reflex-path is not completely drawn. The spinal afferent pupillo-dilatation path only shows the path for pain sensation; the path of the other sensory ways (touch, etc.) which go through the nuclei of Goll and Burdach is only a sketch in its beginning. Of the cranial afferent dilatation paths only part of the trigeminus is drawn. The path of the acusticus, etc., is omitted in order to simplify the scheme.

ILLUSTRATING DR. LUTZ'S ARTICLE ON "THE LIGHT PUPILLARY REFLEX, ITS PATH, AND ITS ABOLITION CALLED IMMOBILITY OF THE PUPIL TO THE LIGHT REFLEX, AND REPORT OF A CASE OF UNILATERAL ARGYLL-ROBERTSON PUPIL, IN WHICH CONSENSUAL REACTION EXISTED IN BOTH EYES."



go through rami comunicantes, ganglion stellatum, troncus cervicalis, to

3d. Ganglion cells of the ganglion cervicalis supremum,

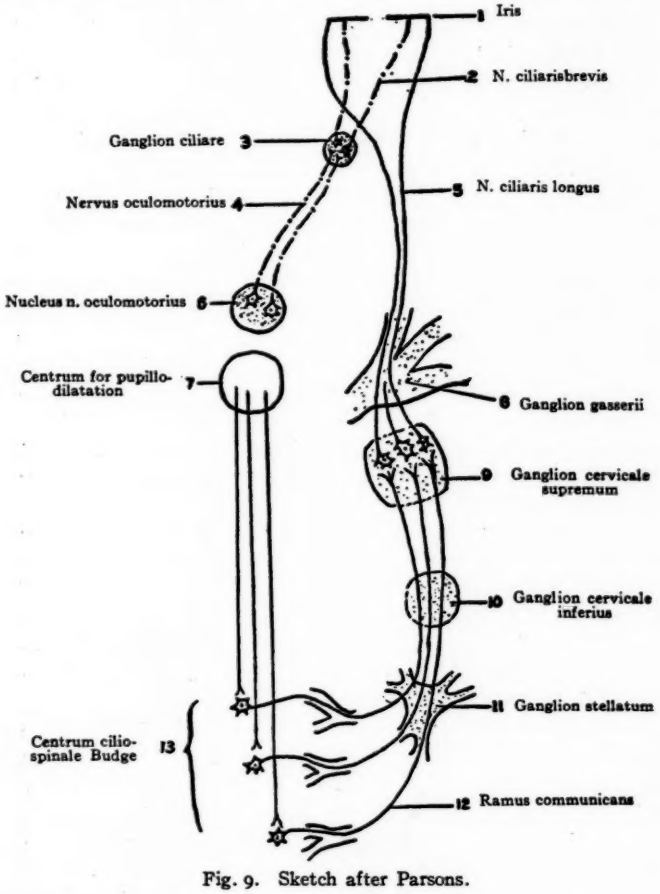


Fig. 9. Sketch after Parsons.

whose fibers run through the anterior branch of the cervico-Gasserian strand and the nervus ophthalmicus to the long ciliary nerves.

4th. Muscle cells of the musculus dilatator iridis.

III.

THE TERMINAL ORGAN, THE IRIS.

After what we have demonstrated it is easy to understand that the terminal organ is under the constant influence of two antagonistic muscles: the sphincter and the dilatator muscles of the iris. During the light reflex we can observe not only the rapid, clonic, and isolated contraction of each of these muscles, but we can also see that the two antagonists are, as all muscles innervated by a cerebrospinal center, under the constant influence of a tonus, which is simultaneous for both eyes. Bach showed that pupillary dilatation through pain continues after section of the trigeminus, sympathicus, and ganglion cervicale superius, and disappears only after section of the oculomotorius. The influence of the two antagonists is even seen after death. In agony, the pupil is large, then it becomes small through the rigor mortis of the sphincter, which is stronger than the dilatator; later on, the pupil becomes large through action of the dilatator which survives longer, and at last, after death of all these muscles, there takes place another slight contraction of the pupil.

But not only are the nervous impulses responsible for this tonus; we have also to consider the elasticity of the tissue and the influence of the vasomotor nerves on the blood vessels of the iris known since the experimental injection of the blood vessels by Primelles (1840): diminution of the tension in the vessels, with less blood in them, produces dilatation of the pupil; increase of their tension, contraction of the pupil, through the radial structure of the iris. In this direction, the ganglion ciliare is of the utmost importance. Lapersonne tells us that we have to consider it at the same time as a cerebrospinal and as a sympathetic ganglion. It has no influence on the exterior muscles of the eye, and cannot be made responsible for irido-dilatation. It can only have influence on the ciliary muscle and on the sphincter. We know that burning of the cornea and iridectomy is followed by degeneration in the large ganglion cells of the ciliary ganglion, and we know from the experiments of Langendorf that even a certain time after death when excitation of the nervous oculomotorius is without effect

on the exterior muscles of the eye electrical excitation of the sensitive ciliary nerves produces irido-contraction. We can therefore divide the pupillary reflexes according to Vennemann into three classes:

1st. Peripheric reflex or vasomotor arc (sensitive nerves of the eye, large ganglion cells of the ciliary ganglion, ganglion cells of the ganglion Gasserii, short ciliary nerves).

(*a*) Hyperfunction shows miosis.

e.g., in Angina phlegmonosa Vincentii, if soft palate is affected (nervus palatinus; nervus maxillaris superior; ganglion Meckeli; ganglion ciliare; nervuli ciliaris breves).

(*b*) Hypofunction shows mydriasis.

e.g., through glaucomatous compression of the nervi ciliaris.

2d. Basal reflex, or light reflex arc.

(*a*) Hyperfunction shows miosis.

e.g., through strong illumination.

(*b*) Hypofunction shows mydriasis.

e.g. darkness, amblyopia.

3d. Cortical reflex arc.

(*a*) Accommodation.

(*aa*) Hyperfunction shows miosis, *e.g.* hyperopia.

(*bb*) Hypofunction shows mydriasis, *e.g.* myopia.

(*b*) Psychical function, attention, etc.

(*aa*) Hyperfunction shows mydriasis.

e. g., fright, strong attention, mania.

(*bb*) Hypofunction shows miosis.

e. g. newly born child, old people, idiocy.

The first one of the three reflexes is unilateral; the two others are always bilateral, consensual, and direct. The first two are always inconscient and involuntary; the last one is not conscient but can be influenced voluntarily.

IV.

PHARMACOLOGY OF THE PATHS OF THE LIGHT REFLEX.

We divide the nerves, from the pharmacological standpoint, into two groups:

1. Animal nerves: they leave the brain or the cord in an

uninterrupted series and proceed directly to the terminal organ. They innerve the voluntary organs.

2. Vegetative nerves: they leave brain and cord only from circumscribed areas; they never go directly to the terminal organs but always enter in their path in connection with some peripheric ganglion. They can pass through different ganglia, but find the real nervous connection in only one of them; they go to the organs that work automatically. They are subdivided into two groups:

a. Parasympathetic nerves: they go direct from the brain to the peripheric ganglia. The parasympathetic nerve of the mesencephalon is the nervus oculomotorius.

b. Sympathetic nerves: they leave the cord through the white rami communicantes, enter the troncus sympathetitus, leave it as gray rami communicantes, and join the spinal nerves.

This division is important for the susceptibility of certain poisons on the nerves, *e. g.* nicotine is a poison for all vegetative nerves, but it never attacks them in their path, but only in the place where they enter into real nervous connection with the last neuron. It is therefore the best means of determining the terminal ganglia of all the vegetative nerves, the parasympathetic as well as the sympathetic nerves. On the other hand, adrenaline excites only the terminal fibers of the sympathetic; it never acts on parasympathetic nerves. The same action which adrenaline has on the sympathetic termination takes place with physostigmine and muscarine on the parasympathetic nerve terminals. The parasympathetic terminals which physostigmine and muscarine excite, are paralyzed by atropine or curare. Meanwhile, muscarine excites the parasympathetic nerves peripherically, picrotoxine and camphor excite parasympathetic nerves only in their central part.

Considering these pharmacological effects and remembering the above-named physiological experiments, we find:

A. Dilatation of the Pupil.

1. Through central paralysis of the sphincter centrum, *e. g.* in asphyxia or botulismus.

2. Through central irritation of the dilatator center, *e. g.* in fear, pain, concentration of thought, etc.

3. Through local application of nicotine to the ganglion ciliare.

4. Through peripheric paralysis of the parasympathetic nervous terminals by atropine.

5. Through peripheric excitement of the sympathetic nervous terminals by adrenaline.

B. Contraction of the Pupil.

1. Through central paralysis of the inhibitions which dilate the pupil, *e. g.* in slight narcosis which eliminates all sensitive and sensorial irritations or in morphine narcosis.

2. Through central irritation of the sphincter centrum by picrotoxine.

3. Through local application of nicotine to the ganglion cervicale superius.

4. Through peripheric excitement of the parasympathetic nerves by eserine.

The afferent sensory pupillo-motoric path could only be influenced by strychnine, which facilitates the transmission of the stimulus from one neuron to the other.

V.

THE ABOLITION OF THE LIGHT REFLEX (CALLED "ARGYLL-ROBERTSON PHENOMENON").

The movements of the pupil can be altered in many different ways, but the most interesting changes are condensed under the name of ARGYLL-ROBERTSON'S PHENOMENON. This abnormal reaction of the pupil is called after its first describer, who published (1869) some cases of diseases of the spinal cord. Basing his conclusions on five patients, he describes the phenomenon as consisting of the following different symptoms: the pupil does not react on light incidence; it reacts very well on convergence and the pupil shows a marked miosis. These observations were confirmed soon afterwards by H. Knapp. Argyll-Robertson was right in so far as further investigation showed the utmost importance of this pupillary reaction for the whole pathology, which is perhaps best illustrated by the

investigations of Mann, who found the Argyll-Robertson phenomenon in tabes even more constant than the Westphal phenomenon. But he was not right in describing the miosis as an integral part. Uhthoff (1914) tells us that miosis is found in only about 30% of the cases; that the others may lack miosis, or can even show mydriasis. Erb had already expressed, long before, the opinion that miosis and immobility of the pupil to light are two different symptoms caused by two different processes. Miosis is found more in tabes, mydriasis more in paralysis generalis. Wilbrand-Saenger called attention later on to a certain atrophy of the iris, which is found from time to time together with immobility to light, and which Dupuy-Dutemps (1905) believes so characteristic that according to him it alone permits the diagnosis of the Argyll-Robertson phenomenon. Finally, Bach stated that together with immobility to light, there is mostly found an abolition of the pupillo-dilatation reflex through excitation of the trigeminus. The phenomenon is called to-day by most authors "immobility of the pupil to the light reflex," and its modern definition is given by Axenfeld in the following terms: "We speak of pure immobility of the pupil to the light reflex, when we find in eyes whose vision is intact, the pupils immovable to light incidence in the same as well as in the fellow eye; during convergence the pupils become miotic, and that in a very marked manner; these eyes lack almost regularly the reflex dilatation through pain or psychic irritation." Winaver Bronislas expresses himself in the same way, but adds a restriction: "Under the condition that there exists no lesion in the eyeball or in the optic nerve."

The importance of the immobility of the pupil to the light reflex lies in its connection with certain diseases which are the consequence of syphilitic infection. It is the special merit of Babinski to have pointed this out. It has never been found in healthy persons, *e. g.* Charpentier and Etchevery investigated 1100 soldiers without finding a single case. We also find absence of the light reflex generally with mydriasis in certain abnormal stages, such as in epileptic attacks, hysterical crises, syncope, catatonic stupor, extreme fright, and excessive muscular consumption; but it is not certain that all these cases are genuine cases of immobility of the pupil to the light reflex. They can be explained as cases of spastic mydriasis. Doubt-

less, we find the typical Argyll-Robertson's phenomenon very exceptionally in certain non-syphilitic diseases, which are: sclerosis multiplex; syringomyelia; atrophia muscularis spinalis progressiva; neuritis interstitialis hypertrophica; ataxia hereditaria; alcoholismus chronicus gravis; and diabetes; oftener, in certain traumatisms of the skull. But these are all rare exceptions, and Grasset (1912) is right when he expresses the importance of the immobility of the pupil to the light reflex for the practitioner in the following terms: "The Argyll-Robertson phenomenon indicates tabes, sometimes general paralysis, always previous infection with syphilis." If we exclude the above-named exceptions where syphilis could not be detected even through the most modern methods of investigations of the blood and cerebrospinal fluid, and even in autopsies, we find immobility of the pupil to the light reflex as a consequence of syphilitic infection in first line in tabes: in incipient tabes it is found in 45% of the cases; in the fully developed, in 85%; in second line, we find it in paralysis generalis—in 17% of the cases in adolescent, and in 50% of them in adults; finally, we find it in about 10% of cases of cerebral syphilis (*e. g.* syndrome of Babinski). The immobility of the pupil to the light reflex can precede a metasyphilitic disease for several years, and can be during that time the only sign of syphilitic infection. It develops always very slowly and after having appeared it, as a rule, remains permanently. But there are also known some rare cases (*e. g.* Eichhorst) which show intermittence, which means that the immobility of the pupil to the light reflex disappeared and reappeared, even several times, under treatment, or without it. It can also develop as a sequel of a paralysis of the nervus oculomotorius in persons where no other sign of tabes or paralysis generalis could be observed. Intermittence is found in all forms, but is mostly observed in cases of cerebral syphilis. The Argyll-Robertson pupil is found with preference in the later stages of syphilis; but it can also appear exceptionally in the first period of the infection: Sulzer (1901), *e.g.*, observed a case about one year after the appearance of the chancre.

The immobility of the pupil to the light reflex is found almost always in both eyes, even when there exists a marked anisocoria, miosis on one side and mydriasis on the other. In rare cases

the phenomenon is found on only one side, and these unilateral cases of Argyll-Robertson's pupil are mostly precursors of general paralysis. All authors agree that a unilateral immobility of the pupil to the light reflex has absolutely the same prognostic significance as its bilateral form—which means that a man with a unilateral Argyll-Robertson is never a healthy man, and is probably a candidate for a severe affection of his nervous system. It is therefore of the utmost importance to make in every case of anisocoria the examination of the light reflex. The unilateral form is very rare. I have tried to collect all the publications on unilateral cases found in literature, and have added them to the end of this article. Its rareness is perhaps best illustrated by the fact that in 1900, in a scientific session in Paris, where Babinski presented a case of unilateral Argyll-Robertson, men such as Déjerine and Gilbert Ballet declared having each seen only one case in their lives. Bach saw one unilateral case during several years as the only symptom of an incipient tabes. Sauvineau saw one case disappear under treatment with mercury. Rothmann, Abelsdorf, and Frenkel-Garipuy saw one unilateral case as a sequel to an improved case of oculomotor palsy. Tanzi saw in a 50-year-old patient with beginning dementia paralytica a unilateral Argyll-Robertson complicated with miosis appear and completely disappear five times during the same year. Unilateral Argyll-Robertson is mostly found in dementia paralytica where we see it in about 3 to 5% of all cases with immobility of the pupil to the light reflex. In the other diseases it is observed less often, and it is natural that it is found relatively oftener in traumatic cases, *e. g.* Krüger, Axenfeld, Weste. In trauma with compression on the brain it can also appear as a transitory phenomenon with loss of consciousness, and it is then to be considered as a very bad prognostic sign. It is also worth while mentioning the experience of Dufour who saw a woman with a slight Westphal, anisocoria, and unilateral Argyll-Robertson, and no other symptom from the nervous system; the patient died accidentally soon afterward, and the autopsy showed all the typical lesions of the whole spinal cord which we are accustomed to find in tabes incipiens.

Grasset gives the following definition of unilateral immobility of the pupil to the light reflex: "The affected pupil re-

mains immovable to light incidence in the same as well as in the fellow eye; the healthy pupil reacts to light incidence in the healthy eye as well as in the affected one." This means that in the affected eye the direct light reflex and the consensual one are abolished. It represents the common form of unilateral Argyll-Robertson. But there are some other observations, as those of Babinski, Bechherew, Berger, Jessop, and Piltz, which show the same peculiarity as my own observation which gave occasion to this study, namely, that the direct light reflex is abolished alone and that the consensual light reflex is found present in both eyes. Such cases represent therefore a pure direct light immobility and the common form of Argyll-Robertson represents a total light immobility (direct plus consensual). One could conclude from this distinction between pure and total light immobility, that we could also find the bilateral form of pure light immobility, that is to say, cases where on both sides the direct light reflex is abolished, but not the consensual one. I do not know if there exist any publications bearing out this point. It seems to me impossible with regard to the above-named description of the last step of the afferent pupillo-motor path. But we must remember how easily we forget to detect the presence of the consensual reaction in bilateral forms of Argyll-Robertson. On the other hand, the above-mentioned sketches give us the explanation that it is theoretically possible to find cases where in both eyes the direct light reflex is present and in both eyes the consensual reaction is abolished. This shows once more how important it is to always make the examination on consensual reaction.

Personal Observation. Mr. J. P., 51 years old, merchant, Spaniard, was sent to my office by his family physician, Dr. C. Desvernine, for an anisocoria which was observed accidentally. His father had died from Bright's disease; he had never had syphilis; his mother died of diabetes; one brother died of diabetes before he was 30 years old; two sisters suffer from polyuria, and one sister from hepatic colic. His wife died from diabetes; she had one abortion, no children. The patient has never been ill and denies absolutely any syphilitic infection; Wassermann reaction of the blood is negative. He consulted his physician for a slight paresthesia in his right hand. He had always shown a peculiar character, and in later times has held some very queer

ideas, with a certain mixture of delirium as to wealth. Upon his wife's death he wished to erect a mausoleum in which a modern bathroom and toilet should be installed, as well as an expensive harmonium.

STATUS: 8—IX—1916. Paresthesia in the right hand, diminution of the patellar reflex on both sides. Vision: R. & L. Eye $\frac{4.5}{14.0}$ plus 2.25 = $\frac{4.5}{4.5}$. Media quite transparent, fundi normal. No diplopia. Marked anisocoria. No ptosis, no enophthalmus, no alteration of the vasomotor innervation. Right pupil: 1.5*mm*, Left pupil: 4.5*mm*. Left pupil: reacts directly on light incidence; consensually on light incidence; on convergence; on accommodation; and on excitation of the trigeminus. Right pupil: absolutely no reaction on direct light incidence; doubtful (consensual) on incidence in the other eye; very well on accommodation; very well on convergence; absolutely no reaction on excitation of the trigeminus.

14—X—1916. Dark room: R. P. : L. P. : 1.5 : 3.5*mm*. Ordinary illumination: R. P. : L. P. : 2.0 : 4.5. Reaction on trigeminus abolished on the left eye.

20—XII—1916. Patient had some mercury injection. Dark room: R. P. : L. P. : 2.0 : 3.0. Ordinary illumination: R. P. : L. P. : 2.5 : 4.0. Anisocoria diminished.

Trigeminus reaction reappeared in the left eye, absolutely clear. In the right eye it cannot yet be detected with certainty. The right pupil reacts slightly consensually on incidence in the left eye, and after staying for a while in the dark room, one can also see a very slight direct light reaction in the right eye.

15—III—1917. Dark room: R. P. : L. P. : 2.5 : 3.5. Ordinary illumination: R. P. : L. P. : 3.0 : 4.5. Left pupil reacts: On direct light incidence; consensually, on light incidence, on the other eye; on convergence; on accommodation. The reaction on trigeminus cannot exactly be detected. R. P. reacts: Doubtfully on direct light incidence; markedly consensually on incidence in the other eye; very well on convergence and accommodation. Trigeminus reflex cannot be detected with certainty.

19—IV—1917. Status: *idem*.

24—X—1917. Status: absolutely the same. Consequently, the patient showed during one year the abolition of the direct light reflex in one eye, whilst the consensual light reaction could be detected in both eyes.

The unilateral cases of Argyll-Robertson have their special and utmost importance in the study of the anatomical founda-

tion of the immobility of the pupil to the light reflex. To explain this, let us consider the different theories and opinions which tend to fix precisely the situation of the anatomical lesion. Different authors have located the lesions in:

(a) *The spinal cord* (efferent pupillo-dilatation path). It was originally the idea of Argyll-Robertson that the cause of this phenomenon was located in the spinal cord. He based his opinion in the miosis and in the fact that the pupils dilate so slowly on applications of mydriatics. This theory, especially defended by Rieger, Forster, and Reichardt, is even sustained to-day (1912) by Siebert and Harald. They base their opinion on the belief that it cannot be solely by chance that we almost regularly find degenerations there. In favor of their opinion we find especially the case of Remak, where there existed besides, an atrophia muscularis spinalis progressiva.

Against this theory we have to remember that miosis is not typical for immobility of the pupil to the light reflex and that interruption of the neuron which precedes the centrum ciliospinale, and which is followed by miosis, can never explain the abolition of the light reflex. Furthermore, we must remember that Déjerine could not find any alteration in the spinal cord in cases with very marked bilateral Argyll-Robertson.

(b) *Ganglion ciliare* (efferent pupillo-contraction path). This idea of Van Gehuchten has been especially defended by Marina and Lafon. Marina bases his opinion on bibliographic and anatomical studies which convinced him that there were many cases of Argyll-Robertson in which no changes were found in the region of the pupillo-motor centers, and further, on his anatomical investigations in the ganglion ciliare which showed him that in any case of immobility of the pupil to the light reflex, degenerations could be found in the ganglion cells of the ganglion ciliare, and that these degenerations were absent in people with normal pupil reaction. Especially one case of unilateral Argyll-Robertson, in which degenerations were only found on the unhealthy side and not in the healthy one, convinced him in this respect. Immobility of the pupil to the light reflex would therefore, according to this theory, be a flaccid paralysis of the efferent pupillo-contraction path, and one could also be tempted to cite in favor of this theory the iris atrophy described by Dupuy-Dutemps.

Against this opinion we must bear in mind that paralysis of the ganglion ciliare with nicotine is followed by pupillo-dilatation and complete paralysis of the pupil. The action of nicotine shows very clearly that we cannot consider the fibers for accommodation as animal fibers of the nervus oculomotorius crossing the ganglion ciliare without entering there into communication with the ganglion cells, and consider on the other hand the light fibers as vegetative fibers of the nervus oculomotorius which do not go directly to the iris, but enter in their route into communication with the short ganglion cells of the ciliary ganglion. Nicotine conduces to paralysis of all efferent fibers of the ciliary ganglion, and it therefore does not explain why the fibers of the light reaction do not work, while the fibers of convergence and accommodation act very efficiently in cases of Argyll-Robertson pupil.

Lesions of the short ganglion cells of the ciliary ganglion could not explain either why we find miosis in about 30% of the cases; in others we find normal pupils or even mydriasis. Paralysis of the short ganglion cells must be followed by dilatation of the pupil.

We have further seen that in some unilateral cases the consensual reaction can be found in both eyes, and I think it more natural to accept that the same efferent fibers conduct the excitation of the direct as well as of the consensual light reflex.

We cannot either consider the iris atrophy of Wilbrand-Saenger as favorable to a flaccid palsy—because Dupuy-Dutemps observed that the atrophy can precede the apparition of the immobility to light.

A lesion in the ciliary ganglion would not explain, either, the abolition of the dilatation reflex following excitation of the trigeminus, which we find so often together with immobility to light, because the efferent pupillo-dilatation path does not enter in nervous connection with the ganglion cells of the ciliary ganglion as shown by the nicotine experiments of Langley and Anderson.

As a rule, we find the Argyll-Robertson bilateral; only exceptionally is it unilateral. This argues likewise against the opinion of Marina. For, if he were right, unilateral Argyll-Robertson should be found oftener than the bilateral, because

we must admit that there is more probability of only one ciliary ganglion being affected.

Against the opinion of Marina we have also to mention the investigations of Andre Thomas (1910) who made autopsies in three cases of Argyll-Robertson; he could not find any degeneration in the short ciliary nerves nor in the ciliary ganglion, nor in the roots which the ciliary ganglion receives from the oculomotorius and the trigeminus.

The fact that we find the immobility of the pupil to the light reflex as a rule bilaterally and only exceptionally unilaterally is also a reason why we cannot expect to find the anatomical substratum of the Argyll-Robertson in the nervus oculomotorius (efferent route) or in the retinæ, nervi optici, corpora quadrigemina anteriora (sensory afferent route). It forces us to search for the lesion in the immediate neighborhood of the centers.

(c.) *Centers:*

1. Fascicular lesions. It was Heddæus who first thought that Argyll-Robertson was produced by a fascicular lesion of the efferent fibers of the photomotor center. He based his opinion on the cases with mydriatic light immobility of the pupil.

Against this opinion we find miosis in about 30% of the cases. Interruption of the efferent fibers is followed by pupillo-dilatation.

Against his opinion we find further the few cases in which the consensual reaction was found in both eyes. As there does not exist a decussation of the efferent fibers, a fascicular lesion nearly always abolishes the direct and the consensual light reflexes at the same time.

2. Nuclear lesions. The opinion that Argyll-Robertson is produced by a nuclear lesion has been defended in first line by Levinsohn.

The fact that precisely characterizes the immobility of the pupil to the light reflex, namely, that the pupil can be contracted voluntarily very well in convergence and accommodation, is an argument against this opinion. We know further that nuclear lesion is followed as a rule by mydriasis.

3. Supra-nuclear lesions. Since pupillo-dilatation is only lacking in light reflex but not in convergence or accommodation

reflex, we have to look for the interruption where the light excitation has not yet reached the centers for pupillo-contraction and pupillo-dilatation. This speaks in favor of a lesion in the communication between the corpus quadrigeminum anterius and the photomotor centers. This opinion has been especially defended by Landois, Déjerine, v. Monakow, and Uhthoff. Our considerations as to the path of the light reflex showed us that an interruption of the communication between the corpus quadrigeminum and the nucleus photomotoricus must be followed by abolition of the direct and consensual light reflexes, and will remain unilateral if the lesion remains unilateral; but as these neurons run each very near the other in the neighborhood of the third ventricle, we can also understand that even small lesions must abolish the light reflex in both eyes. In favor of this opinion may be cited: the cases of syringomyelia complicated with unilateral Argyll-Robertson; the traumatic case of Pope who found at autopsy the third ventricle full of blood; the traumatic cases of Guillain, Rochon-Duvigneaud, and Trosier. Furthermore, anatomical investigations of many authors who found in the gray matter itself, around the third ventricle, degenerations of the fibers, blood extravasations, and obstruction of the vessels. In favor of this localization we have, furthermore, the case of Moeli (*Archiv f. Psychiatrie*, xviii.), who found the lesion of a bilateral Argyll (with intact vision, intact mobility of the eyeballs, and good convergence reflex) to be a tumor involving both sides of the posterior walls of the third ventricle. Von Monakow (*Archiv f. Psychiatrie*) found in a case of one unilateral loss of the pupillary light reflex a small sclerosed focus in the tissue between sphincter nucleus and the external geniculate body. Such a localization of the anatomical lesion would also agree closely with the fact that tabes is preferably a disease of the afferent sensitive routes. Paralysis and tabes are clearly characterized by the early degenerations of the last termination of the sensitive nerves.

This theory would also better explain how the communication of the sensory-afferent way and the efferent pupillo-dilatation way is interrupted in both eyes. For we must remember that in cases of Argyll-Robertson there is not only loss of the influence of light in the pupil, but also of the influ-

ence of the sensory excitations through trigeminus and the psychical excitations through the cortex. From anatomical researches and clinical observations in cases of hemiplegia Déjerine deduced that the sympathic path descends to the mesencephalon. We can therefore say that the lesion which cuts the communication between sensory afferent path and pupillo-contraction center cuts at the same time the communication between the sensory afferent path and the sympathicus of the mesencephalon.

One could be tempted to say that the miosis argues against a lesion in this last afferent sensory neuron. This argument is eliminated by the fact that amaurosis of both eyes, *e. g.*, through atrophia optica tabetica is without any influence on the form of the light immobile pupil, that is, the miotic pupil remains miotic.

Lafon (1909) called attention to the importance of studying the modifications of the Argyll-Robertson pupil consequent upon lesions of the different parts of the afferent and efferent paths of the light reflex way. These are:

I. Arcus Sensorio-Motoricus.

(a) Lesion of the afferent sensory path, *e. g.*, amaurosis is without any effect on the pupil.

(b) Lesion of the efferent pupillo-contraction path:

1. The action of atropine is retarded.

2. Compression of the post-ganglionic fibers, *e. g.*, by glaucoma, produces dilatation of the pupil.

3. Paralysis of the oculomotorius also produces a dilatation of the pupil, but not so strong as in uncomplicated cases.

II. Arcus Sensitivo-Motoricus.

We do not yet know the influence of destruction of the trigeminus or of the ganglion cervicalis superius.

III. Arcus Corticalis.

(a) It is true that abnormal excitations of the cortex as we see them, *e. g.*, in maniacal people, are without influence on the Argyll-Robertson pupil.

(b) We do not yet know the influence of the hypofunction of the cortex on the Argyll-Robertson, *e. g.*, in sleep, in syncope, or narcosis, that is, if the pupil grows smaller.

From this observation we can conclude that the lesion of the afferent pupillo-contraction path is without influence; that the

lesion of the cortical inhibition path is probably also without influence; that we do not know anything certain about the influence of the afferent and efferent pupillo-dilatation paths; and that finally lesions on the efferent pupillo-contraction path have doubtless an influence on the Argyll-Robertson pupil. We need further observations in this direction.

Erb holds that light immobility and miosis are two different symptoms produced by two different processes. But it seems to me more natural to accept that the appearance of miosis depends more on the extension of the lesion. We can accept that miosis is produced by the lesion of the mesencephalic sympathicus or by the interruption of the last sensory afferent neuron with this mesencephalic sympathic. This miosis differs fundamentally from Horner's syndrome in that it lacks ptosis, enophthalmus, and alterations of the vasomotor innervation, but we can consider that this difference is caused by the fact that in Argyll-Robertson pupil the neuron is interrupted before the centrum cilio-spinale, and that in Horner's the same path is interrupted in the neuron whose origin is found in the ganglion cells of the centrum cilio-spinale. Levinsohn (1908) has especially studied the phenomenon of miosis; he called attention to the fact that in Horner's syndrome, miosis disappears in a dark room, notwithstanding the existence of a paralysis of the sympathetic, and that in Argyll-Robertson pupil, darkness is without any influence, which is easily understood by the complication of light immobility and which best shows that miosis and light immobility are two different things. Furthermore, miosis is found very much more pronounced in the Argyll-Robertson pupil than in Horner's syndrome. We cannot explain the miosis by a lesion of the afferent sensitivo-motor path, because we find many cases with marked miosis where the sensibility is entirely normal, and on the other hand many cases without miosis where the sensibility is abolished. The unilateral cases of Argyll-Robertson show that we cannot localize the cause in the afferent sensory route (retina; nervus opticus; corpus quadrigeminum), because such lesions should conduce to bilateral miosis. As, furthermore, the fibers of pupillo-contraction for light incidence and for accommodation as well as convergence are intimately intermixed in the nervus oculo-

motorius, we cannot expect to discover the cause of the miosis in the efferent pupillo-contraction path. These arguments induced Levinsohn to search for the lesion in the nucleus, and he admits that in the nucleus sphincter there are two parts: one which is responsible for the pupillo-contraction on light incidence, and another for pupillo-contraction through accommodation, convergence, etc.; if the first part is destroyed, we have immobility of the pupil to the light reflex; and through irritation of the second part miosis; if the destruction extends to the other part, we have total immobility of the pupil with mydriasis. Against this theory we should remember that miosis can persist for several years and it would be very improbable that during this whole time we would have miosis through pure irritation of the area; further, if the miosis were produced by irritation, it should always be complicated with spasm of the accommodation (musculus ciliaris). This latter has never been observed, and we see therefore, that the study of the miosis also forces us to search for the lesion not in the centers but in their neighborhood, that is, in that region where the afferent path joins with the efferent pupillo-dilatation path. An interruption of the connection of the afferent sensitive pupillo-dilatation path with the photomotor center explains only the loss of the trigeminus dilatation reflex. I think that the marked miosis is produced by the interruption of the fibra descendens fascic. longit. post., which goes from the corp. quadrig. ant. to the centrum cilio-spinale. If the lesion destroys the crossing of Meynert (Meynert's fontänenartige Haubenkreuzung) just in the median line, it produces bilateral miosis, if it acts only on one side, unilateral miosis follows. If the lesion is to be found there, it would explain at the same time why we find so often light immobility and miosis combined. A lesion of the fibra descendens fascic. longit. post. explains also the presence of miosis in diseases of the spinal cord, without that light immobility is found at the same time. This leads me to believe that the light immobility is produced by interruption of the fibers (of the fascic. tecto-bulbaris) which go from the corpus quadrig. to the nucleus photomotoricus and the miosis by the interruption of the fibers (of the fasciculus tecto-spinalis) which go from the corp. quadrig. anterius to the centrum cilio-spinale.

With regard to the foregoing considerations on the route of the light reflex and with regard to the various clinical observations and pathological discoveries in cases of immobility of the pupil to the light reflex, I think that we cannot accept the argument of some authors, that the Argyll-Robertson phenomenon can be produced in different ways by anatomical lesions in different parts of the light reflex path. I think that the lesion must always be found in the stage between the corp. quadrig. and the nucleus principalis lateralis.

SUMMARY.

A. Description of the light reflex:

1. The light reflex consists in a contraction of both pupils on increase of illumination, and in bilateral pupillary dilatation on reduction of illumination. Only light incidence on the retina reduces contraction of the pupil, while irritation of all other cranial and spinal nerves is followed by irido-dilatation.

2. The adaptation of the retina is of great importance for the amplitude of the pupillary reaction. Perhaps the amacrine cells of the centrifugal neuron chain are responsible for this.

3. The size of the pupil is in inverse proportion to the square root of the light intensity (Ovio).

4. The light reflex begins at the end of the fifth fœtal month and is not abolished, in healthy persons, through age.

B. Path of the light reflex:

1. The light strikes the cones and the rods of the whole retina. The pupillo-motor effect of it is stronger in the macular region, and decreases rapidly towards the periphery. The multi-stratified bipolar cells of the second retinal neuron conduct the light irritation from different cones and rods to the multi-stratified ganglion cells of the third retinal neuron. The fibers of these latter cells conduct the light stimulus in the thick fibers through opticus, chiasm, where they perhaps do not undergo the semi-decussation of the visual fibers, and through the tractus opticus. Hemikinesis cannot be regarded as proof of the semi-decussation of the pupillo-motoric fibers in the chiasm. In front of the corpus geniculatum laterale the pupillary fibers enter the brachium conjunctivum and go through these to the ganglion cells in the deeper layers of the

corpus quadrigeminum anterius. The fibers of these last cells reach, through fasciculus tecto-bulbaris, the nucleus photomotoricus; part of them keep on the same side and produce the direct light reaction; collaterals of them go on the other side and produce the consensual light reaction. These central semi-decussations take place in the neighborhood of the nucleus photomotoricus.

2. As *center* can be regarded the frontal pole of the nucleus lateralis principalis (von Monakow). But, it seems to me more natural to regard as pupillo-motoric center the deeper layers of the corpus quadrig. anterius, from which originate the two efferent pupillo-motor paths: firstly, for active pupillo-contraction, through nucleus principalis lateralis, and secondly, for active pupillo-dilatation through the centrum cilio-spinale.

3. The *efferent pupillo-contraction path* begins in the nucleus oculomotorius. The fibers of the photomotor center enter without crossing the nervus oculomotorius, and reach, through the short root, the small ganglion cells of the ciliary ganglion. The fibers of these latter reach, through the short ciliary nerves, the musculus sphincter iridis.

4. The *pupillo-dilatation* is produced by irritation of all cranial or spinal nerves except opticus, and by central inhibition. Its irritation produces inhibition of the photomotor center (responsible for pupillo-contraction) through the ascending fibers of the fasciculus longitudinalis posterior, and produces on the other side active pupillo-dilatation through the descending fibers of the fasciculus longitudinalis posterior. These latter fibers reach the centrum cilio-spinalis in the spinal cord. Its fibers go without crossing through the ventral roots of the last cervical and the first two thoracic nerves as rami-comunicantes to the truncus sympathicus cervicalis and to the ganglion cervicale supremum. The fibers of this ganglion reach, through the anterior branch of the Gasserian strand, the ophthalmic nerve and the long ciliary nerves, the musculus dilatator iridis. These latter fibers have no connection with the ciliary ganglion.

5. The iris stands under a constant tonus of the nervus oculomotorius as well as of the nervus sympathicus (long ciliary nerves). It is furthermore constantly influenced by the

large ganglion cells of the ciliary ganglion, which received stimuli from the Gasserian ganglion (radix longa) and the plexus sympathicus carotideus (radix sympathica) or vasomotor branch of the truncus sympathicus cervicalis.

6. The light reflex—the direct as well as the consensual one—is a basal reflex, and does not go through the ciliary ganglion.

C. Immobility of the pupil to the light reflex:

1. The Argyll-Robertson phenomenon consists in the abolition of the direct and consensual pupillary reaction to light incidence in both eyes, while the reaction on convergence is found normal. It is mostly connected with abolition of the pupillo-dilatation reflex on irritation of the trigeminus. In 30% of the cases we find also miosis.

2. It is one of the most important indications of diseases following syphilitic infection, and it can also be found exceptionally in other diseases. In rare cases the Argyll-Robertson phenomenon is found only on one side. The affected eye then shows no light reaction: its pupil does not contract on incidence of light in the same as well as in the fellow eye.

3. To explain the phenomenon we have not to look for the lesion in the spinal cord, nor in the ciliary ganglion, but in the region of the photomotor center: the interruption of the connection between the last afferent sensory step and the photomotor center explains the immobility of the pupil to light incidence; if the lesion is large enough it interrupts the connection between fasciculus tecto-bulbaris and nucleus principalis lateralis on both sides; otherwise it cuts it only on one side. Minor lesions only interrupt the connection of the direct light reaction path and leave untouched the collaterals going to the other side; in such cases the consensual reaction is found present in both eyes.

Miosis can be explained by the interruption of the first neuron for active pupillo-dilatation (fibra descendens fasciculi tecto-spinalis).

The loss of the trigeminus reflex can be explained by the interruption of the connection between fibra ascendens fasciculis longitudinalis post. (trigeminal part) and the photomotor center; the lesion eliminates the inhibitive influence of the afferent pupillo-dilatation path.

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CORRIGENDA

I have to correct the opinion (expressed in the first part of this paper) that the crossing of the pupillary fibers in the chiasm has not yet been proven. The semi-decussation of the pupillary fibers in the chiasm has been demonstrated anatomically in cats by S. Ramón y Cajal, and in man by Van Gehuchten. Histological researches of the brachium conjunctivum ad corp. quad. ant. showed that this consists of two different bundles: 1. One which comes down from the lenticular region and probably therefore from the cortex, the fasciculus cortico-bigeminalis; it ends in descending lateral dendrites in the deepest layer of the corp. quad. ant. 2. The other comes from the optic tract and ends in ascending dendrites in the next layer higher up. Ramón y Cajal could demonstrate that the latter receives the majority of the fibers from the opposite eye; Van Gehuchten believes that two thirds of all pupillary fibers cross the chiasm. These anatomical findings fully explain the experiments of Trendelenburg in cats and especially the very exact observation in man by Pierre Marie and Chatelin.

If we ask ourselves the reason why hemikinesis can be observed far more easily in animal experiment, we must remember that the lower animals have no visual cortex. Corp. geniculatum externum, pulvinar and calcarine fissure become important only in mammalia. On the other hand, the corp. quad. ant. have in the lower animals a far more complicated structure, and are so large that anatomists speak of a tectum opticum. This leads us to the conclusion that the pupillomotor function of the corpus quad. ant. in man is not a function specifically different from that of the visual cortex,—but only the most primitive or phylogenetic oldest form of the visual function. Therefore, we can no more hold the opinion that the fibers of the radiation of Gratiolet have no pupillo-

motor effect; on the contrary physiological experiments have shown that electrical excitation of the calcarine fissure produces a marked miosis. As a consequence we shall find hemikinesis not only in lesions of the frontal visual neuron (optic tract) but also in those of the occipital neurons. This explains to us the observations of hemikinesis in hemianopsia posterior made by Clifford B. Walker and Déjerine. Furthermore considering the fact that in man the corp. quad. ant. are only very small compared with other visual centers, we can understand why hemikinesis through lesions of one optic tract is far more difficult to observe in man than in animals, because the clinical appearance of the hemiopic pupillary reaction is hidden by the action of the pupillo-motor fibers which descend from the opposite hemisphere and partially cross with the fibers of the fasciculus tecto-bulbaris. This consideration may explain to us, furthermore, why have we not definite clinical observations of hemikinesis without hemianopsia, such as we can expect from a lesion of the brachium conjunctivum. Such an occurrence would be concealed through the action of the pupillo-motor fibers descending from both cortices.

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For war reasons I could not get the following most important monographs about pupils:

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BUMKE: *Pupillenstoerungen.*

WILBRANDT-SAENGER: *Neurologie des Auges.*

THE INFLUENCE OF FUNCTION UPON THE STRUCTURE OF THE EYE.

By Dr. EDUARD UHLENHUTH, New York.

(From the Laboratories of The Rockefeller Institute for Medical Research.)

Author's Abstract.)

IF an eye of a larva of the European fire salamander (Salamandra maculosa) is severed from the optic center and is grafted, with a flap of surrounding skin, to another larva of the same species, the retina of the grafted eye undergoes a more or less severe degeneration, but after a short time begins to regenerate and the normal condition of the retina may be reestablished completely. In the most severe cases the entire retina is destroyed with the exception of the most peripheral parts. The nuclei are broken down and form large masses of detritus which fill the space of the destroyed retina. The most interesting phenomenon during this process of degeneration is the behavior of the cells of the retina pigment epithelium. The pigment epithelial cells detach themselves from the pigment epithelium and migrate freely into the detritus masses; here they assume a phagocytic function and it is due mainly to these cells that the masses of detritus are quickly removed from the eye. Their activity lasts, although in a considerably diminished degree, even during the process of regeneration.

Regeneration is brought about mainly by mitotic divisions in the area of the retina next to the destroyed part; mitotic divisions are found in the inner and outer granular layers as well as in the ganglionic layer. Only after very mild degener-

[1] Abstract of paper read before Section of Ophthalmology, New York Academy of Medicine, Feb. 18, 1918. The complete paper will appear in the *Biological Bulletin*.

ation the loss of the few degenerated cells is compensated by mitotic divisions of the cells of the pars retinæ ciliaris only. In the beginning the part regenerated in place of the destroyed area of the retina is an undifferentiated mass of cells. Later on an epithelial-like granular layer is found which by regeneration of the fibrous layers from the old part into the new part is differentiated into the inner and outer granular layers and into the ganglionic layer. Finally the rods and cones regenerate from the outermost cells of the outer granular layer. At this time the activity of the pigment cells is stopped entirely.

However completely the structures of the grafted eyes may be restored, these eyes are unfit to function since no stimuli can be transmitted to the brain; for the grafted eyes are severed from the visual centers. Since despite this lack of function the functional parts of the eye, viz.: the cell elements of the retina, the rods and cones, which in regard to the specific function of the eye appear to be the most highly specialized parts of the eye, and in many cases the nervous structures may remain normal, it is clear that the opinion of many anatomists and surgeons that the structures of an organ cannot regenerate without the organ being in active function is erroneous. The structure of the grafted eyes not only regenerates, but also persists without the aid of function. Eyes preserved three and a half years after having been grafted show all the functional elements of the retina present.

Moreover, the same result can be obtained, if the functional stimulus, viz.: any trace of any kind of light is prevented from reaching the grafted eyes. Two curves are shown which demonstrate that the retinæ recovered from their state of degeneration without light and secondly regenerated in darkness just as rapidly as in light.

Since the grafted eyes are severed from the nerve centers, the experiments also prove that a trophic stimulus, exerted by the nerve centers upon the tissue, is not a factor involved in the regeneration and maintenance of the functional structures of the eye.

It can be shown by a number of facts that the degeneration of the grafted eye is due to the interruption of the blood circulation; the restoration of the normal conditions in several parts of the eye can be watched with a binocular and it is

observed that degeneration stops and regeneration begins as soon as the newly established blood circulation reaches the part in question. Degeneration and regeneration in the grafted eyes are matters of nutrition.

The phenomena exhibited by the cells of the pigment epithelium are such as are known to occur in retinitis pigmentosa. Many of the grafted eyes show a very considerable retinitis pigmentosa, caused by the migration of the pigment cells into the retina and there many of these cells break down and deposit their pigment. The progress of the retinitis pigmentosa can be watched in the grafted eyes from its very beginning. The conclusions drawn from the facts presented are the following: retinitis pigmentosa is the cause of a disease; it follows the degeneration of the retina. Since the migrating pigment epithelial cells tend to remove the products of degeneration by their phagocytic action, it is a defensive mechanism against the disturbance. In no case, neither in the grafted eye nor in fragments of tissue transferred to a culture medium was there ever observed a migration of the pigment epithelial cells except the pigment epithelium was freed from the adjoining layer of the retina, either by degeneration of this layer or by detachment of the retina. It seems, then, that the pigment epithelial cells must be freed from the rods and cones; otherwise they are not able to migrate.

The facts mentioned in this paper were illustrated by twenty-two lantern slides and by a living specimen of the American tiger salamander (Amblystoma tigrinum) to which an eye had been grafted from another animal of the same species eight months before demonstration; the eye was in a normal condition so far as could be judged from its appearance.

THE OPERATIVE TREATMENT OF TRAUMATIC COLOBOMA OF THE LID.

By Professor L. v. BLASKOVICS, Budapest.

Translated from *Arch. f. Augenheilk.*, vol. lxxxi., 1916.

(With five figures in the text.)

THE most frequent war injury to the eyelids is the coloboma of the eyelid. The cases usually come to us in the cicatricial stage. While these injuries occur during peace times, they are so uncommon that no typical operation has been developed. The principle for the reconstruction of the eyelid has remained unchanged and consists in reproducing that condition which was present directly after the injury and then to suture the wound in such a way that the lid returns as much as possible to its normal position. This principle cannot always be obtained, as the injuries consist not only in tears of the lids but often defects which must be replaced by fresh tissue. Hence the operative procedure is necessarily an atypical one. Nevertheless the frequency of these cases in war times has developed definite types of operation which more or less follow the two following varieties:

I. In these cases the eyelid is torn away from the canthus, usually the inner one. A scar is present which runs downward from the angle of the lids, varying in length. The lid is anchored fast in an ectropionated position. The difficulty of suturing the eyelid to its original position is given by the absence of a sufficient raw surface at the canthus whereby a proper union of the wound edges and a satisfactory adhesion of the eyelid in its original position can be obtained. Under these conditions the eyelid, though it is in its normal position

at the conclusion of the operation, through renewed contraction the ectropion returns. In order to correct this defect, it has seemed advisable to fasten the lid to the canthus in a way which can be described as hanging the lid upon a small flap or spur.

I find that von Pflugk has designed a similar procedure and has obtained good results with it. The method is extremely simple and consists of the following steps:

(*a*) The scar is outlined by two parallel running incisions which are joined at their extremities. After thoroughly excis-

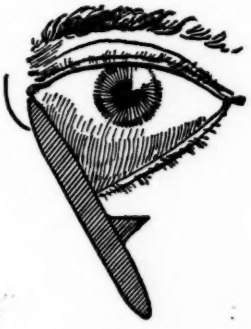

FIG. 1.

ing the scar tissue as well as the lateral strands, the lower lid is perfectly free.

(*b*) At the inner margin of the wound a flap is formed with its base at the level of the angle of the lids (Fig. 1).

(*c*) At the temporal margin of the wound just below the adherent margin of the lid a straight incision is made into the skin. This small incision gapes in the form of a triangle and serves to receive the little flap which has just been described in *b*.

(*d*) The wound edges are carefully united (Fig. 2). The little spur-like flap fits smoothly into the triangular gaping wound just described. The remainder of the wound can be sutured with a continuous suture. The recovery in my cases has been unusually smooth. The new adhesion of the lid was

always sufficiently strong and the spur-like flap gave a large enough raw surface to unite the lid to. The spur itself becomes a part of the skin of the lid. No relapse whatever has been observed.

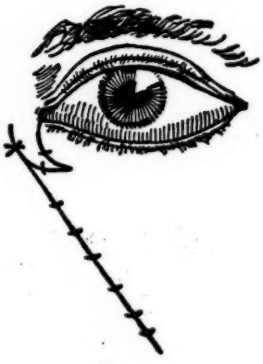

FIG. 2.

This operation is as well suited to the upper lid as to the lower.

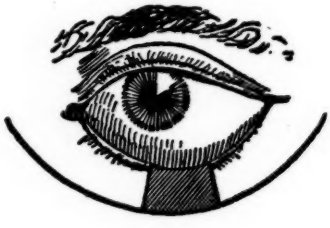

FIG. 3.

II. The second procedure is suited for those uncommon colobomata of the lower lid in which the margin of the lid has suffered traumatism in its middle part. After cicatrization of these wounds at the line of separation of the lids there is a cicatricial area between the two margins of the lid and the lid itself is ectropionated.

(*a*) The first incision is made at about 1*cm* below and parallel with the margin of the lid and throughout the whole length of the lid (Fig. 3).

(*b*) The scar is then circumscribed with two incisions which run vertical to the margin of the lid and completely separate the bridging flap.

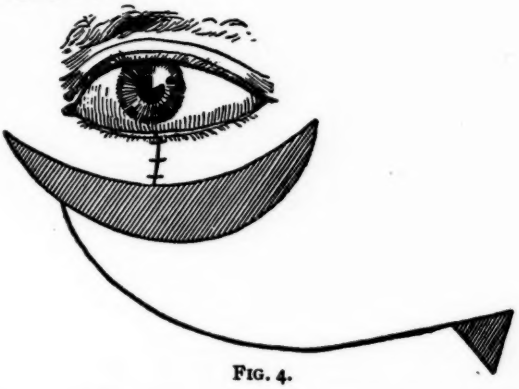

FIG. 4.

(*c*) The scar is thoroughly dissected and its lateral expansions are excised.

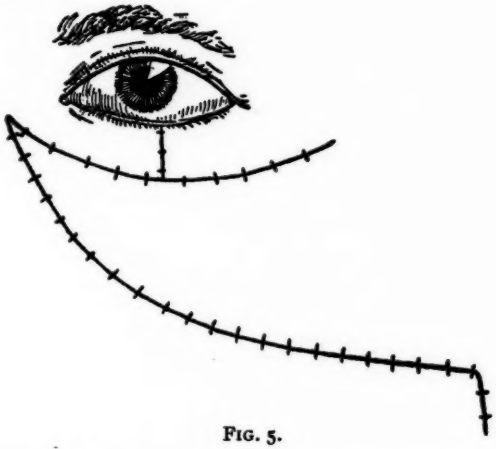

FIG. 5.

(*d*) The margins of the coloboma are then approximated by three interrupted sutures. The ectropion is thereby corrected and a semi-lunar raw surface is formed underneath the sutured lateral flap (Fig. 4).

(*e*) To cover this wound surface another curved incision is made running from the lower margin toward the temple. The apex of this new-formed flap is 1½ to *2cm* to the temporal side of the inner canthus. The lower extremity is considerably further out than the level of the outer canthus.

(*f*) After detaching this flap a triangular piece of skin and subcutaneous fat is excised from its lower margin. The base is directed up and measures 1½*cm*.

(*g*) The columns of this triangle are first united. The flap is thereby displaced toward the nose and covers the defect by occupying a broader surface of raw area than before. The flap is then sutured with a continuous suture (Fig. 5). The results of this procedure were just as good as the first one. The flaps unite, owing to their broad base, without any trouble. The position of the lid is normal. Relapses were not observed.

OPTICO-CILIARY NEUROTOMY, RESECTION OF THE OPTIC NERVE. A SUBSTITUTE FOR ENUCLEATION.

By Dr. A. H. PAGENSTECHER, Wiesbaden.

Translated from *Archiv f. Augenheilkunde*, vol. lxxix., 1915.

THIS operation, which is of course an old one, has in recent years been performed quite frequently at our hospital. It results in furnishing a good stump for a moveable glass eye, in case the latter is at all necessary. From 1898 to 1913 this operation has been performed more than 100 times in our eye hospital. In most of these cases the indication was not to shield the eye from the danger of sympathetic inflammation, but to preserve the blind eye if it was entirely free from danger to the other eye, from the standpoint that a blind eye was always better than an artificial one.

This resection of the optic nerve is indicated in my opinion in two groups of cases. The first are the cases of absolute glaucoma with great pain. The second those of total staphyloma where the staphylomatous formation has not advanced to the point of causing a great deformity. In my experience resection is followed by a reduction of the intraocular tension, the pain in the glaucomatous eye is relieved, the glaucomatous process in the eye with the staphyloma is arrested. An artificial eye is usually not necessary. If the patient for cosmetic reasons desires an improvement in his appearance, a thin glass shell can be made to fit over the affected eye, thus giving much better cosmetic results than with an ordinary artificial eye. The patient moreover is not required to wear his artificial eye constantly.

A few other cases remain of eye injury where the danger of

sympathetic inflammation could not be definitely excluded, but where enucleation was not concurred in. The resection is then practiced of a large piece of the optic nerve, believing that hereby in many cases a definite prevention of sympathetic inflammation is obtained. The eyeball after this operation shrinks, particularly if the injury has been a penetrating one, and in all circumstances forms an excellent cushion for the artificial eye. In my experience resection of the optic nerve in absolute glaucoma or in total staphyloma has never led to an atrophy of the eyeball, as is believed by many.

Our method of operating is as follows, which agrees entirely with the method as first advocated by my father, except that the resection of the muscles is no longer thought necessary. The conjunctiva is divided between the inferior and internal recti muscles about 1*cm* distant from the corneal limbus. The scissors then proceeds into the deeper parts along the sclera. With a double hook introduced in the scleral tissue posterior to the equator in a meridian which connects the corneal diameter with the optic nerve entrance, the eye is drawn upwards and outwards, keeping traction exactly in this meridian. This puts the optic nerve on stretch and with strong scissors curved on the flat, the optic nerve is divided well back of its insertion into the eyeball. Keeping up the traction with the hook, the optic nerve remaining attached to the eyeball is then brought forward on the flat of the scissors until it appears in the wound. The optic nerve is then seized with a toothed forceps and the nerve is divided close to the sclera. All of the ciliary nerves about the optic nerve entrance are then divided. The eyeball is allowed to return to its normal position and it is very important at this moment to begin to exercise strong pressure upon the eyeball in order to prevent a retroöcular hemorrhage. The conjunctival wound is closed with one or two sutures and a firm pressure bandage applied. The after treatment is not longer than the one following simple enucleation, if no pronounced bleeding has taken place.

I hope this short description of the operation will correct the impression, which seems to be general, that this operation is a difficult one, and in the near future I shall subject the cases which have been operated on by this method to a careful reëxamination, which I shall then be glad to publish.

REVIEW

R. FOSTER MOORE. **The Retinitis of Arteriosclerosis, and its Relation to Renal Retinitis and to Cerebral Vascular Disease.** *Quarterly Journal of Medicine*, vol. 10, nos. 37 and 38, October, 1916, January, 1917.

The one hundred and ten cases which form the material for this study were examined and kept under observation as long as was possible at a general hospital and at an eye hospital. The cases were all subjects of general arteriosclerosis and were divided into two groups: (1) those who came to the eye hospital (Moorfields) because of impairment of sight due to vascular disease which had involved the retina, and (2) another group of patients at a general hospital (St. Bartholomew's) who were suffering from the results of cerebro-vascular disease and in whom the eye changes had given rise to no symptoms.

The author confirms the signs of retinal arteriosclerosis which were first pointed out by Marcus Gunn, and believes that obstruction of the blood-flow in the veins where they are crossed by the arteries is worthy of special emphasis, and is the most valuable and important single sign of severe arteriosclerosis. This phenomenon at the arterio-venous crossings is never seen apart from arteriosclerosis and is always present in sclerosis of any considerable degree. The variations in the degree form a reliable means of judging the extent of the sclerosis. The author has drawn attention to the displacement of the lines of the vein at these crossings which he thinks is a very striking and important symptom. He considers this phenomenon under two heads: The evidence of obstruction to the flow in the vein and displacement of the line of the vein. The distension which a vein peripheric to the crossing shows is generally not marked and is not an indication of the degree of obstruction. When a vein is

crossed twice by the same artery the trapped portion of the vein is very much reduced in size and when a vein crosses over a sclerosed artery an actual narrowing of its lumen can be seen. If an artery crosses a vein at an angle in the early stages of sclerosis the line of the vein will remain unaltered; as the thickening of the arterial wall increases, the line becomes diverted in proportion to the degree of the sclerosis. In other words, as a vein and an artery converge, the vein at first is diverted so as to lie alongside of the artery; it then passes under the artery and on the other side again runs alongside of the artery for a short course. This same phenomenon the author points out occurs when a vein crosses over an artery.

Retinal hemorrhages, the author believes, are directly due to the impairment in the nutrition of the vessel wall and to changes in the blood. The hemorrhages are not necessarily related to hyperpiesis on account of the following reasons: (1) pressure in the retinal arteries is not increased in arteriosclerosis; (2) a marked increase in the number of hemorrhages is not infrequent before death when there is no increase of blood pressure; (3) the retinal hemorrhages are very frequent in the various anemias in which there surely is no increase of blood pressure.

Under the heading of Arterial Disease as a Cause of Optic Atrophy, the author states that atrophy of the optic nerve either partial or complete may be brought about in two ways as a result of arteriosclerosis: first by thrombosis of a diseased artery with sudden loss of sight and subsequent optic atrophy, or the sclerosis of the arteries may slowly reach such a stage that they are no longer able to transmit sufficient blood to the tissues. This latter group is a particularly interesting one. The histories of eight patients are given, in whom partial atrophy followed from this cause. They were all the subjects of severe arteriosclerosis with marked disease of the retinal vessels. The loss of sight gradually developed in the course of several weeks or months. To the ophthalmoscope disease of the retinal arteries is well-marked; there may also be a little œdema of the disk. The fields show irregular constriction. The author agrees with Nettleship and Gunn, that retinal arteriosclerosis is more frequent in the female sex.

A very important and interesting part of this article is devoted to the discussion of Arteriosclerotic Retinitis as a clinical entity. In this, retinal exudates occur in eyes with retinal arteriosclerosis differing in appearance from renal retinitis and sufficiently distinctive for the author to set up a new disease, arteriosclerotic retinitis. In this condition there is disease of the small vessels. There are well-marked signs of retinal arteriosclerosis and retinal hemorrhages are present. The retinal exudates which are characteristic occur in the form of small white dots, spots, or small areas. They are irregularly circular in outline, vary in size, not larger generally than the diameter of a retinal vein. There is no œdema and no pigmentary disturbance. The dots sometimes seem to be in relation to the branches of the smallest veins, in other cases they have no definite relationship. It is common to find a group of them between the macula and the disk, and sometimes they constitute a complete or a partial star-figure around the macula. These dots are slow to develop and are very slow to undergo any changes, though the author has observed that they may completely disappear without leaving any trace. In the advanced cases there are some larger plaques which are identical with these discrete dots and never present any œdema, hemorrhage, or pigmentary changes. The arteries sometimes show a white plaque-like deposit in their perivascular sheaths. This is entirely different from the ordinary sheathing of the vessels, as the blood stream is not affected and the plaques may completely disappear.

Arteriosclerotic retinitis is often present in only one eye. A characteristic is the scantiness of the white dots, their arrangement, their course, and that they are always associated with severe arteriosclerosis of the retinal vessels. The usual exudates and hemorrhages seen in renal and diabetic retinitis are always absent. The diagnosis as to life in these cases is very uncertain, as they often live a number of years. The cause of death then is a disease of the vascular system and not one of the kidney.

A. K.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By JOHN M. WHEELER, M.D., Secretary.

MONDAY EVENING, FEBRUARY 18, 1918. MARTIN COHEN, MD., CHAIRMAN.

Dr. M. Uribe-Troncoso presented a case of **chorioretinitis with fixed opacities in the vitreous.** A man 35 years old had a marked chorioretinitis in the left eye. The usual sumptoms of cloudiness of the vitreous, large floating opacities, some exudates on the retina, papilla congested, and hazy and tortuous veins were manifest, but on looking carefully with direct ophthalmoscope and +7. or +8. in the sight hole, a curious aspect was detected. In the middle part of the vitreous, nearer the upper and inner side, appeared numerous small, gray-black, round, sharply outlined specks, mostly about 1*mm* in diameter, diffusely distributed and sometimes arranged geometrically in squares or constellations. By shifting the mirror in such a manner that only the edge of the beam of light falls upon the specks, they take a whitish transparent color with darker centers. These specks are stationary, as can be proved by focusing one group and having the patient's eye moved up and down and again fix. They are studded in one membrane, whose edge is visible on one side, and keep their arrangement and reciprocal distance on all movements. The anterior segment of the eye is normal. Vision $\frac{20}{100}$. In the right eye vision $\frac{20}{30}$. The fundus looks at first glance normal, but the vitreous is slightly hazy. With +7. a group of specks similar to the other eye, but less

numerous and of lighter color, can be detected. They are also fixed, but no sign of membrane or liquefaction of the vitreous could be observed.

The significance of these fixed opacities is interesting. Straub and Dhai have mentioned similar appearances which they call fixed vitreous dust and compare to the deposits on Descemet's membrane. They think they are heaps of leucocytes strewn on a membrane, which always form the wall of a cavity containing liquefied vitreous. The single leucocytes being too small cannot be detected by the ophthalmoscope and only produce a light haziness of the vitreous. They remain always isolated in the substance of the vitreous, never forming groups, which are only present on the walls of the cavities. The dust has been considered for a long time as a characteristic symptom of syphilitic chorioretinitis, still Dhai in fourteen cases found only one of true syphilitic origin and eight of tuberculous.

In Dr. Uribe-Troncoso's case although the left eye had certainly a liquefied vitreous and a membrane existed in which the heaps of leucocytes were fixed, in the right eye the condition seems different because the specks are not studded on a membrane on the wall of a cavity, as Straub believes, but were probably fixed by the coagulation of the fibrin in the serous exudate produced by the beginning diffuse inflammation. The question may also arise if these opacities do not represent the proliferation of connective tissue cells of the fibrillary stroma of the vitreous, made apparent by the inflammation on the knobs of the mesh; still its appearance is highly suggestive of heaps of leucocytes, similar to deposits on Descemet's membrane.

Dr. J. W. WHITE presented a case of **spasm of accommodation** (a full report appears in this issue).

Dr. WHITE showed also a case of **obliterating endarteritis,** probably tuberculous in origin. L. N., aged 20, developed sudden blindness in left eye over night two months ago. No history of illness. Vision R. $\frac{20}{15}$; L. eccentric vision only, confined to a sector in the upper field. The fundus showed an obliterating endarteritis of the superior temporal and nasal vessels, the retina in the corresponding part of the eye ground showed deep opacities and hemorrhages. Wassermann nega-

tive. Tuberculin tests gave a slight febrile reaction but a decided focal reaction in fundus.

Dr. DUANE presented for Dr. S. T. HUBBARD a case showing **congenital retraction movements.** The patient is a Hebrew girl 16 years of age. She tilts her head to the left. When she attempts to look to the left the left eye shows limited abduction. When she attempts to look to the right (adduction) the left eye turns up and rotates inward, and it recedes into the orbit, allowing narrowing of the palpebral fissure. The patient has myopic astigmatism, but the vision is normal in each eye with the correction.

DISCUSSION. Dr. JULIUS WOLFF: I am interested in Dr. Hubbard's case of retraction movements of the eyeball because I was the first in this country to describe the clinical entity or syndrome of which the retraction is one symptom. My article appeared in the ARCHIVES OF OPHTHALMOLOGY, May, 1900. I here reported and analyzed five cases and emphasized the opinion that these cases were not so very infrequent but that the retraction of the eyeball had been overlooked. Dr. Herman Knapp told me at the time my cases were presented at this Section that he had not seen a case before, but in the above number of the ARCHIVES he also published a case.

If I may read the summary of my article it will be found to cover all the points brought out by Dr. Duane this evening.

"Retraction movements in the human eye have been described in only seven cases, to which five are herewith added. The retraction movements never occur as a solitary symptom, but always form part of the same group of clinical symptoms, producing a well-defined clinical picture, whose characteristics are as follows: The condition is always congenital. Retraction occurs during attempted adduction, which may be absent or present, but is always less than normal. Retraction is accompanied by narrowing of the palpebral fissure. Partial or complete paralysis of the external rectus of the retracted eye is regularly present. Some cases present a moderate constant retraction and narrowing of the fissure even in the primary position. In these cases attempts at abduction produce a propulsion of the globe and widening of the fissure. When the retraction is considerable the cornea is turned upwards in some cases, downwards in others, even when the fellow

eye makes a purely lateral movement. This is due probably to resistance made by the optic nerve.

"Two explanations are offered to account for the retraction: the *faulty insertion* and the *fixation* theories. Though the former is supported by some evidence, the latter accounts more satisfactorily for all the symptoms and is even better supported by evidence. Surgical interference may benefit some of the cases. There is reason to believe that the retraction movements are often overlooked and that these cases are not so rare as the small number reported would indicate."

Dr. Wolff showed a model illustrating the mechanism by which the retraction is brought about.

Dr. ARTHUR J. HERZIG presented a case of **organization of lens matter in the capsule,** the center having been absorbed. This shows as a dense annular ring, creamy white in color and apparently well organized, situated behind the iris in the position of the capsule. The patient, age 39, white, was struck in the left eye by a stone at the age of twelve. Iridectomy was performed six months afterward. The eye never gave the patient any trouble until December 27, 1917—twenty-seven years afterward, when he fell downstairs striking his eye with the handle of a coal scuttle which he was carrying at the time. The eye became inflamed and only local remedies were applied at the time until the eye became painful and he presented himself at Dr. Cutler's clinic at St. Luke's Hospital where I first saw him. Wassermann and Von Pirquet tests are negative. The eye is only slightly painful and all inflammatory signs have disappeared. Vision is nil in the injured eye, and normal in the other. As long as the eye is quiescent the speaker will not remove it. The case is presented to show the annular character of the organization of lens matter with the center of the lens absorbed.

Dr. GEO. H. BELL presented a case of **pulsating exophthalmos** due to a fracture at the base of the skull, in a man aged 46. Patient fell off a load of lumber about six weeks ago, striking on the back of his head. At that time patient bled from nose, mouth, and ears and was unconscious only a few minutes. Also he vomited some blood. Ten days ago he came to Dr. Bell's clinic at the New York Eye and Ear Infirmary complaining of headache, buzzing noises in ear and head. There was

much proptosis and marked restriction of the movements of the globe. When the eyeballs are pressed back into the orbit, you can see and feel the pulsation. Patient complains of diplopia. There is an audible bruit in both temporal regions, more pronounced on the right side. Pressure on common carotid on right side stops the bruit. There is an engorgement of the retinal veins in both eyes and some chorioretinitis in right. V. O. D. $\frac{20}{50}$, V. O. S. $\frac{20}{40}$. The congestion of conjunctiva has increased. The exophthalmos in right eye is 24 and in the left 22. X-ray plate showed diseased ethmoids and frontals, but no fracture could be made out. This is a case of an aneurism of internal carotid in the cavernous sinus. Blood pressure 120. Urine normal.

Dr. Bell expects to have a general surgeon ligate the common carotid on right side, and will make a report later on as to the result of the operation.

Dr. JOHN M. WHEELER presented a case of **paralysis of divergence.** The patient is a man (W. J.) 25 years old. August 1, 1917, while digging a ditch his vision suddenly became confused, and since that time he has seen double at a distance. He was in a hospital three months but left unimproved. His vision is normal in each eye, and there is no abnormal condition of the interior of either eye. Pupillary reactions are normal. The urine is normal. Wassermann tests of both blood and spinal fluid gave negative results. Neurological examination was negative. The characteristic features of divergence paralysis are well shown. The tropometer shows normal excursions of each eye in all directions. The far point for binocular single vision by approximation is about 14 inches, while the far point by recession is 24 inches. The point of equilibrium is 4 inches from the root of the nose. Diplopia decreases slightly as the patient looks to the right or to the left, whereas it would increase in looking in one of these directions if there were a paralysis of the external rectus. In this case paralysis of divergence probably resulted from hemorrhage into the divergence center, while the patient was under physical exertion.

DISCUSSION by Dr. CUTLER: I have seen two cases similar to the case reported. In one, a boy of ten, paralysis of the hypothetical center accompanied an attack of poliomyelitis

in which certain muscles of the neck and shoulders were involved. It was noticed as the boy recovered from the severe depression of the acute stage of the disease and remained stationary for a considerable time. The patient was taken to Boston later for treatment of the after-effects of the poliomyelitis, and I have not heard whether recovery of the function of the ocular muscles took place.

In another case, a woman with syphilis, the diplopia was typical and there was in addition a slight exophthalmos and tenderness on pressure of the left eye, as if there had been an involvement of orbital tissue, which I think was quite apart from the condition causing the diplopia, which was very characteristic of these cases. This symptom diminished very gradually under intensive treatment of the constitutional disease, but finally disappeared entirely.

In neither of these cases was the diplopia distressing. In the former the boy had considerable myopia and was able with his glasses quite readily to ignore one image or the other. In the second case there was moderate hypermetropia and the patient was more comfortable without glasses for distance.

In one other case at least, that I have seen, the diplopia was a distressing symptom and necessitated the closure of one eye, as prisms, base out, were not a satisfactory relief.

DISCUSSION by Dr. ALGER: Dr. Wheeler's case seems an absolutely typical one of divergence paralysis. My attention was first attracted to the condition by Dr. Duane's original paper to which reference has been made. Shortly afterward my first patient, a very intelligent gentlemen, appeared and his description of his symptoms was enough to suggest the diagnosis. From time to time I have seen others, and in the *Transactions* of the American Ophthalmological Society of 1916 reported nine original cases.

I do not believe the condition can be anything like as rare as the very small number of reported cases would imply. The diagnosis is easy enough. It is simply overlooked because there is no reference to the subject in most neurological or ophthalmological text-books, and most of us do not think of the possibility of an ocular paralysis without limitation of rotation.

Cases like this would predicate the existence of a special

center for divergence, though so far as I know it has not been localized. Granting the presence of centers for divergence and convergence, on clinical grounds, there must, theoretically at least, be others for maintaining single vision in the other associated positions of the eyes. So far illustrative cases have not appeared.

The etiology, course, and prognosis seem to be much like those of the better known paralyses. My cases included probable hemorrhage from advanced angiosclerosis, syphilis, tabes; while one followed diphtheria, and was associated with paralysis of accommodation and deglutition.

Dr. EDUARD UHLENHUTH: **The influence of function on the structure of the eye** (an abstract of this paper is published in this number of the ARCHIVES). The paper was illustrated by many lantern slides and by a living specimen of the American tiger salamander.

MONDAY EVENING, MARCH 18, 1918. MARTIN COHEN, M.D., CHAIRMAN.

Dr. H. H. TYSON reported a case of **electric ophthalmia with chorioretinitis** which had been shown but not reported at the February meeting. "P. S.," age 53 years, visited the Knapp Memorial Eye Clinic on February 12, 1918, stating that his eyes burned and itched with some mucous discharge from the lids following an exposure to an electric flash of 670 volts, which occurred from a short circuit while fixing a motor the night previous. He stated that he had red vision for about ten minutes during which time he was practically blind. Examination of the eyes showed circumcorneal injection of both eyes, more marked in the right eye, with photophobia and lacrimation. Vision R. $\frac{20}{100}$ and L. $\frac{20}{70}$. There was a central scotoma for red and green and metamorphopsia. Fundus right eye showed small œdematous patch in retina with two small linear hemorrhages located about two disk diameters to nasal side of optic disk (direct method) and in the left eye a patch of pigment in the choroid located somewhat in a similar position, as if being the result of previous exposure. The ophthalmoscope findings were similar to those found after direct exposure to sun in viewing solar eclipses. Vision improved later to $\frac{20}{30}$ with correction. Up to 1913

only one case of marked chorioretinitis from the effect of electric light had been published, and that one was by Uhthoff. In May, 1913 (*Zeitschr. f. Augenheilk.*), Paul Knapp reported a second case. A man who had been exposed to a short circuit had vision reduced to one half and had metamorphopsia which later disappeared, and vision improved to normal. P. Knapp agrees with Fuchs as to the cause in these cases being the luminous rays, because they affect chiefly the choroid, pigment epithelium, and exterior layers of the retina, while the ultraviolet rays injure mostly the inner retinal layers. This case added to the literature makes the total three with chorioretinal changes.

The case was discussed by Dr. DENIG who had seen a similar case in which there was typical orbital neuralgia from short circuit.

Dr. A. D. MITTENDORF said that he had seen a case of chorioretinitis from bright lights in the Arctic region.

Dr. L. W. CRIGLER presented a case of **chronic simple glaucoma** in a young man, age 21, with a hypermetropia of 12 D. His father and mother are first cousins. They are living and healthy and have good vision. There is marked contraction of the visual fields. Vision is reduced to $\frac{20}{200}$ in the right eye, and less than $\frac{20}{200}$ in the left. The fundus showed the usual picture of chronic glaucoma. Tension in right and left eyes, between 30 and 35*mm* of Hg.

Dr. CRIGLER presented a case of **aphakia in a man 48 years of age.** The patient gives a reliable history of having had a cataract in his left eye twelve years ago. He does not recall having had an injury. On examination the eye presented the typical signs of a dislocated lens: the anterior chamber was deep, the iris was tremulous, the pupil was round, somewhat larger than that of its fellow, and responded feebly to light. Intraocular tension measured 30*mm* of mercury. Vision was reduced to hand movements at three feet in a very much contracted temporal field. The fundus could be best seen with a +8 D. lens. There were numerous opacities in the vitreous but the lens, even with the pupil dilated, could not be found. The optic nerve head was cupped and atrophic. The retinal vessels were pushed to the nasal side of the disk. Following the development of the cataract, the lens evidently

became spontaneously dislocated, associated with a tear in its capsular envelope, which in turn produced a secondary glaucoma. Except for the floating opacities in the vitreous the fundus could be easily seen and no evidence of the lens or its nucleus could be found. The right eye is normal.

Dr. GEO. H. BELL presented a case of **bilateral papillo-œdema** (published in full in this issue).

Dr. BRUDER thinks that papillo-œdema in this case was due to kidney disease and not to sinus trouble. Œdema, hemorrhages, and headaches might be caused by nephritis.

Dr. LESZYNSKY does not think that the patient presented symptoms which should have led to the diagnosis of tumor. He related a case presenting headaches, vomiting, and choked disk. Nephritis was present, and recovery ensued. He thinks that papillo-œdema of this type must be unusual in sinusitis.

Dr. GUTTMAN thought that it was unusual for papillo-œdema due to accessory sinus infection to be bilateral. Nephritis may have been caused by infection of the sinuses.

Dr. LAMBERT thinks that the neurologist should have been informed of the presence of nephritis. Recovery from albuminuric retinitis is common. He thinks it impossible to tell the cause from the ophthalmic appearance in papillitis.

Dr. LEVITT thinks that papillo-œdema in this case was not due to sinus disease but to nephritis.

Dr. ARTHUR S. TENNER presented a case of **chancre of the palpebral conjunctiva.** The patient is a young colored woman 22 years of age, married last December. She came to the clinic at the Harlem Hospital, on March 18th, complaining of a painless swelling of the left upper eyelid of two weeks' duration. There is a moderate circumscribed swelling of the lid producing ptosis. The swelling is indurated and extends over the whole upper lid. Neither the skin of the lid nor the lid margin has broken down. The eye is not irritated. On everting the upper lid the tarsus and tarsal conjunctiva are noted to be thickened and reddened with a sharply defined raised border. There are a few spots of dirty gray exudate. There is no pain nor tenderness. The preauricular gland is painlessly enlarged. A scraping of the conjunctiva showed numerous living spirochetæ. Some months ago this patient

had a "stye" in this eye. This stye may have been a chalazion which ruptured and left an opening in the conjunctiva for the entrance of the virus.

One week later a general maculopapular secondary eruption was present covering chest and abdomen and declared by a dermatologist to be of recent origin. The lesion of the lid was smaller and healthier looking and evidently in the terminal stage. No treatment had been used.

Dr. BRUDER said that it probably was not a chancre because the whole tarsus is involved, that it has not the characteristic ulcerated, punched-out appearance of chancre.

Dr. LAMBERT thought that both the appearance and the location argue against chancre. It may be tuberculous.

Dr. AGATSTON called attention to the history of a stye and thinks this may be an irritated chalazion.

Dr. E. M. ALGER presented a case of **pemphigus conjunctivæ.** This patient appeared at the Post-Graduate clinic a few days ago with the following history. She began having trouble with her eyes about three years ago which has steadily grown worse ever since in spite of constant treatment. The diagnosis was apparently made very early, both as to the eyes and the numerous lesions in the mouth, nose, and throat. She has never had any bullæ on the skin. She has, since that time, visited one hospital after another in the desperate hope of getting some relief. The left eye, which was first involved, was finally operated upon by one of our colleagues, who adopted the perfectly logical plan of attempting to prevent the cicatricial obliteration of the conjunctival sacs by extensive skin grafts. The result as we see it was complete failure. There is a complete symblepharon of both lids, while the margins of the lids are attached to each other throughout their entire length by a thick skin which completely covers the cornea, to which it is probably grown fast. The patient is rather bitter over the results of the procedure. As a matter of fact the operation had nothing to do with the final condition because the fellow eye gives every appearance of ending in exactly the same way at no very distant time. It shows a nearly complete obliteration of both conjunctival sacs, and the bridge of skin connecting the lid margins has already advanced far from the external canthus toward the cornea, only a small

part of which remains in sight. It is, however, so far, clear and the vision is good. Projecting into this space from beneath the upper lid margin is a large thick-walled vesicle similar to those on her lips.

Four types of pemphigus are recognized to-day. First, the so-called "essential shrinkage" of the conjunctiva, without vesicles; second, the cases in which lesions occur on the conjunctiva alone; third, cases with conjunctival and associated mucous lesions, and fourth, those associated with classical pemphigus of the skin. The difficulties of positive diagnosis in the first two classes do not need to be pointed out.

Pemphigus is one of the rare skin diseases, and conjunctival pemphigus is far more uncommon. The characteristic blebs may involve the outer layers of conjunctiva, in which case they are of short duration and rarely seen, or they may involve, as in this patient, the entire thickness and last several weeks. Corneal lesions are rarely seen in the vesicular stage. In any case the bullæ finally break down leaving raw surfaces which undergo extreme cicatricial contraction, or where they are opposed to each other, result in symblepharon and finally in obliteration of the conjunctival sacs and total blindness.

Nothing definite is known to-day regarding the cause of the disease. No successful method of treatment has so far been devised for real pemphigus. There is no natural tendency to get well; indeed, the prognosis is bad so far as life itself is concerned, while as to vision it is absolutely bad.

Dr. Denig saw this patient very early in the disease when the diagnosis was not so obvious. In fact he made it ahead of the nose and throat men which speaks well for his clinical acumen.

Dr. DENIG saw this patient about two years ago. She had purulent conjunctivitis and complained of sore throat. A rhinologist reported rhinitis. After treating her for six weeks Dr. Denig noticed adhesions in the lower fornix. He then discovered blebs in the mouth and nose. Adhesions increased and skin grafting was advised. Patient then disappeared.

Dr. LAMBERT saw a case of acute pemphigus with Dr. Huddleson several years ago. It followed submucous resection of the nasal septum and the appearance suggested smallpox. The patient died in four days of general septicæmia.

Dr. CURTIN saw a case of bilateral symblepharon three years ago and thought it trachoma. Later the patient died of pemphigus.

Dr. COHEN said that microscopical examination of the tissue in these cases early shows endarteritis, and later connective tissue cells.

Dr. MAX TALMEY read a paper on **suggestions for improving the cataract operation.** Homer Smith's preliminary capsulotomy with the author's modification always yields a large capsular opening. The latter and thorough separation of the cortex during the interval render possible complete removal of the lens and thereby fulfil the first requirement for a perfect cataract operation, *i.e.*, the attainment of entirely clear media. To obtain the second requirement, a round central pupil, there is only one way that can always be depended upon, and this is to close the wound completely by sutures. This has been tried extensively but all procedures hitherto devised have been imperfect and have caused frequent failure. The author's improved procedure consists in a downward section following capsulotomy after an interval of six hours. Three threads are inserted through the borders of the wound and then the lens is expressed. The middle thread is now tied even if the iris should happen to lie in the wound. After putting the iris in correct position the lateral threads are tied. The sewing of the wound differs essentially from that employed by other surgeons by the number of sutures, the inclusion of a sufficient amount of tissue, and the operative stage at which the threads are inserted. The author believes that these steps do not complicate the cataract extraction but rather facilitate it by rendering it more easy to deal with complications such as prolapse of iris and of vitreous body. This method with omission of capsulotomy would even divest the intracapsular extraction, but it was just the shortcoming of the latter that suggested the above operation. The danger of infection is decidedly enhanced by the sutures and must be obviated by the most scrupulous asepsis and antisepsis.

Dr. LAMBERT said that suturing entails risk to the eye. He thinks he should not feel justified in trying such a difficult feat. He thinks the Homer Smith operation accompanied

with danger, and has had glaucoma following preliminary capsulotomy.

Dr. TALMEY said that he had not had an opportunity to try the technique which his paper suggests but that his ideas were not less valuable because of being purely theoretical.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY FOR THE SECOND, THIRD, AND FOURTH QUARTERS, 1917.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen; W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI, Berlin; H. PAGENSTECHER, Strassburg; K. WESSELY, Würzburg; and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New Haven; CALDERARO, Rome; CAUSÉ, Mayence; CURRAN, Kansas City; DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v. POPPEN, Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S., New Rochelle, N. Y.

(*Continued*)

V.—ANATOMY, EMBRYOLOGY, MALFORMATIONS.

40. DAVIES, D. L. **Anophthalmos and microphthalmia.** *British Journal of Ophthalmology*, July, 1917.

41. MAGITOT, A. **The aqueous humor and its origin.** *Annales d'Oculistique*, cliv., 2, 3, and 4.

DAVIES (40, **Anophthalmos and microphthalmia**) contributes five cases of this anomaly to the literature on the subject. The possible causes are discussed in the paper, and three photographs illustrate the appearances of the patients.

T. HARRISON BUTLER.

MAGITOT (41, **The aqueous humor and its origin**) makes an exhaustive critical review of the entire literature on the subject, and comes to the following conclusions:

(1) The primitive aqueous humor is secreted during fœtal life between the third and fifth month by the neuroglia cells accompanying the hyaloid vessels. These cells disappear after their secretory function has been fulfilled.

(2) The aqueous humor has a triple rôle: (*a*) optic and

static; (*b*) protective for the retinal elements; (*c*) as a conserving medium for the lens.

(3) Its chemical composition is very much similar to that of the cerebro-spinal fluid. It contains no colloids and no nutritive elements for the nervous tissue but it constitutes an excellent medium for the conservation of the energy of this tissue.

(4) Under normal conditions *the aqueous humor is without movement.* Contrary to the universally accepted theory, Magitot affirms that there is no circulating current in the anterior chamber and no drainage into Schlemm's canal. However it is probable, he says, that it is absorbed very slowly. Whatever quantity is absorbed is replaced by the neuroglia cells of the retina: the cells of Müller, those around the ora serrata, and the clear ciliary cells whose prolongations constitute the zonula. The fluid is produced by these cells by a process of dialysis, a phenomenon entirely different from secretion and transudation.

(5) There are two systems of lymphatics in the eye. One is independent of the other. They are to be found in the perivascular sheets. There is no direct communication between the aqueous humor and the lymph of the eyeball.

(6) The fluid reformed after a puncture of the anterior chamber is serum, transuded from the capillaries, and a slight amount of aqueous originating from the vitreous. The paper is a very able critical discussion of the theories advanced, experiments performed, and facts established mostly by Leber's school and contains a complete bibliography of great help to those who are especially interested in this subject.

SCHOENBERG.

VI.—THE SENSE OF SIGHT.

42. CERISE, L. **Two cases of double hemianopsia with conservation of macular vision.** *Archives d'ophtalmologie*, XXXV., 5.

43. EDRIDGE-GREEN, F. W. **The relation of ophthalmology to the theory of vision.** *British Journal of Ophthalmology*, July, 1917.

44. PARSONS, J. H. **The apocritic principle and the evolution of visual perceptions.** *Ibid.*, July, 1917.

45. TRAQUAIR, H. M. **Bitemporal hemiopia: the later stages and the special features of the scotoma.** *Ibid.*, March, 1917.

46. VINSONNEAU. **The macular vision in hemianopics with intact macula.** *Archives d'ophtalmologie*, XXXV., 5.

PARSONS (44, **The apocritic principle and the evolution of visual perceptions**) describes the apocritic principle as a "schematic description of patent facts." It can be analyzed into two consecutive stages: a selective segregation, and a creative synthesis.

This paper does not lend itself to abstraction and should be read in the original. T. HARRISON BUTLER.

EDRIDGE-GREEN'S (43, **The relation of ophthalmology to the theory of vision**) theory of vision may be stated as follows: A ray of light impinging upon the retina liberates the visual purple from the rods and a photograph is formed. The rods are concerned only with the formation and distribution of the visual purple, not with the conveyance of light impulses to the brain. The ends of the cones are stimulated by the photo-chemical decomposition of the visual purple by light, and a visual impulse is set up which is conveyed to the brain by the optic nerve. The character of the stimulus differs according to the wave length of the light causing it. But though the impulses differ in character according to the wave length of the light causing them, the retino-cerebral apparatus is not able to distinguish between the character of adjacent stimuli, not being sufficiently developed for the purpose. The power of discrimination differs in individuals, hence the varieties of color perception which are met with. The object of the paper is to show that every known aberration of function, in accordance with the theory, is represented by a disease of the eye, none of which have hitherto had a satisfactory explanation. Vision being photo-chemical there should be defects in accordance with well-known photo-chemical facts.

The rods having the special function of regulating the photo-chemical sensitiveness of the liquid surrounding the cones, there should be diseases corresponding to aberration of this function.

When the sclerotic, choroid, and the pigment cells of the retina are removed the external fovea is seen as a small basin with four canals leading to it. In normal conditions there is a circulation of lymph sensitized by the visual purple from the surrounding parts of the retina into the external fovea and then out through the choroid into the lymphatic spaces of the optic nerve. This retinal circulation is greatly aided by the

four canals. In cases of optic neuritis this starfish-like arrangements of the canals can be seen with the ophthalmoscope. It can also be seen in high myopia. In one case it was seen red and there was a small black spot at the center. Obstruction of the canal should cause night blindness, and this we find to be the case in myopia.

The paper goes on to consider the causation of nystagmus, amblyopia, erythopsia, night blindness, functional disturbances, reversal of the color fields, and scintillating scotomata.

T. HARRISON BUTLER.

VINSONNEAU (46, **The macular vision in hemianopics with intact macula**) has noticed that patients with hemianopia due to a war injury of the head, with intact central vision and emetropia, have the acuity of vision reduced in the eye situated on the same side with the hemianopsia. In patients with right-sided homonymous hemianopsia the right eye is the poorer, and vice versa. The author called attention to this in 1915, and the fact has been confirmed by a number of other authors. In this paper he reports two more such cases.

SCHOENBERG.

TRAQUAIR'S (45, **Bitemporal hemiopia: the later stages and the special features of the scotoma**) paper runs through three numbers of the Journal. It is well illustrated, and should be consulted by neurologists and ophthalmologists specially interested in disease of the pituitary body, and chiasma.

The author sums up his conclusions as follows:

(1) The perimetric defects in bitemporal hemiopia follow a typical or normal development. Commencing in the upper-outer quadrant, the field is involved in a circular manner, the loss proceeding clock-wise in the right field, and counter clock-wise in the left.

(2) The central defect or scotoma behaves in the same manner.

(3) This type of field defect is due to interference with chiasmal fibers, but is largely independent of the ultimate cause; it occurs in bitemporal hemiopias from a variety of diseases.

(4) The immediate cause is probably a chiasmal neuritis comparable to that which, acting upon the optic nerve, produces a retrobulbar neuritis.

(5) The origin of this chiasmal neuritis is not known.

(6) In tumor cases, and perhaps in some non-tumor cases, mechanical pressure also acts, and mainly in the later stages.

(7) In these later stages the normal type of progress may be "swamped" by pressure effects and greatly altered.

(8) These observations provide evidence from the clinical side in support of the looped arrangement of the chiasmal fibers.

(9) They also show that the papillo-macular bundle forms a little chiasma within the chiasma, and that its fibers are similarly arranged. T. HARRISON BUTLER.

CERISE (42, **Double hemianopsia with conservation of macular vision**) reports two cases of this nature. The first patient sustained an extensive war injury of the occipital region and, after the usual period of blindness, following such accidents, he recovered a vision of $\frac{1}{10}$ O. D.; $\frac{2}{10}$ O. S. The field, doubly hemianopic in each eye, was reduced to the central vision, 10° to 15° around the fixation point. He had also convergence insufficiency, hemiparesis, and difficulty in writing. The author believes that the lesion was bilateral and that an island of cortex in the right occipital lobe from which the fibers go to both maculæ remained untouched. Since the crossed macular fibers are more numerous than the direct ones, the macula of the left eye is the better provided and the vision of this eye is better. The second patient, 74 years old and arterio-sclerotic, had for some time a right homonymous hemianopsia and set up, through some vascular changes, a left-sided hemianopsia. The macula remained intact. Vision O. D. $\frac{1}{10}$; O. S. $\frac{3}{10}$. The field was reduced to 10° around the fixation point. According to the author an island comprising the macular center must have remained intact in the right occipital lobe which preserved the central vision in both eyes. SCHOENBERG.

VII.—REFRACTION AND ACCOMMODATION.

47. NORDENSON, J. W. **On the rules for the correction of astigmatism.** *British Journal of Ophthalmology*, March, 1917.

48. PATON, L. **Functional spasm of accommodation.** *Ibid.*, October, 1917.

49. POST, M. H. **Change of refraction following an attack of diabetes.** *American Journal of Ophthalmology*, November, 1917.

POST (49, **Change of refraction after diabetes**) observed the refraction in a diabetic woman after an acute attack and found an increase of + 0.75 D. Some months later the eyes returned to their former condition. No lens opacities were found. ALLING.

The object of NORDENSON'S (47, **On the rules for the correction of astigmatism**) paper is to answer the question, whether in any given case astigmatism should be corrected or not.

Indications for the correction of astigmatism.

In general correction is needed only if it increases visual acuity. When correction improves acuity it may be advisable on account of the following factors:

(1) The need of accurate detail vision.

(2) Asthenopia: which is of two kinds:

(*a*) Accommodation asthenopia.

(*b*) Eyelid asthenopia. The orbicularis can correct as much as two to three diopters of astigmatism by pressure on the globe when the error is against the rule, but this action causes fatigue.

(3) Dangers to the eye from astigmatism. Astigmatism may favor the development of strabismus and myopia.

Contraindications to correction.

(1) The inconvenience of wearing glasses. If one eye be normal and the other astigmatic it is not generally necessary to order glasses. The exceptions are those cases in which there is asthenopia, or if strabismus is threatening. It may also be useful to correct the error when accurate binocular vision is called for.

(2) Non-improvement of acuity by correction. In this case it is useless to correct, but the author goes on to state what every ophthalmologist must have observed, that the vision of an eye which at first shows no improvement may, after a lapse of time which may extend to months and years, begin to gain in acuity.

(3) *Intolerance of correction.* In many cases the patient will not wear the correction even if accurate. This is especially

noted in elderly people who have never worn glasses. In the case of these older patients it may be impossible to persuade them to persevere with the glasses which cause discomfort.

(4) The author notes that Lagrange is opposed to prescribing glasses for children. This surgeon considers that there is a decided tendency for astigmatism to cure itself, and that this change is inhibited by wearing spectacles. Nordenson regards this hypothesis as unproved. The views advanced by the author will meet with little response in England or America, where the tendency to order an absolutely accurate correction of ametropia is steadily gaining ground. Most ophthalmologists will agree that an uncorrected error of refraction, especially astigmatism, even when no symptoms are evident, must be a cause of an expenditure of brain energy which in the long run tells its tale. In general the cases which do not require correction are those which never consult the oculist.

T. HARRISON BUTLER.

True spasm of accommodation is one of the rarest conditions met with in ophthalmology. Hirschberg, writing in 1884, says: "I scarcely believe in the existence of acute and not at all in the chronic form of accommodative spasm." Hess expresses similar views. True spasm of accommodation may be defined as the sudden development of a high degree of apparent myopia which disappears under atropine. PATON (48, **Functional spasm of accommodation**) cites a case of this nature.

T. HARRISON BUTLER.

VIII.—THE MOTOR APPARATUS.

50. CAMILLE and FROMAGET, H. **Latent nystagmus.** *Annales d'oculistique*, cliii., 11.

51. LEAVITT, M. J. **A case of recurrent paralysis of the right abducens lasting twenty-one years.** *Ophthalmic Record*, September, 1917.

52. MARDELLIS, A. **Paralysis of one abducens due to a fracture of the petrous portion of the temporal bone. Recovery.** *Clinique ophtalmologique*, viii., 3.

53. MENACHO, M. **Associated movements of the upper lid during mastication (the Marcus Gunn symptom).** *Annales d'oculistique*, cliv., 4.

54. ROUSSEAU, F. **The paralysis of the oculomotor nerves during otitis media suppurativa and their complications.** *Ibid.*, cliii., 12.

55. SUKER, G. F. **A new ocular muscle symptom in exophthalmic goiter.** *Journal of the American Medical Association*, April 28, 1917.

56. WOODRUFF, H. W. **Tendon transplantation of the eye muscles.** *Ophthalmic Record*, November, 1917.

LEAVITT'S (51, **Recurrent paralysis of the right abducens lasting twenty-one years**) patient was a woman of 23, who had twenty-five or thirty attacks of paralysis of the external rectus of the right eye, the first coming on during an attack of measles when she was a year and a half old. The attacks lasted usually about four or five weeks while the mobility was normal during the intervals. The probable explanation seems to be that some vascular or œdematous changes take place at the nucleus. ALLING.

MARDELLIS (52, **Paralysis of one abducens due to a fracture of the petrous portion of the temporal bone**) tells us of a patient who was struck on the head by a falling tree and immediately complained of a severe headache, epistaxis, and diplopia. Examination revealed a complete paralysis of the left sixth cranial nerve. The paralysis remained stationary for about four months, when it disappeared suddenly, and the motility of the left eye again became normal. The author then modified his diagnosis of a complete severance of the left abducens, due to a fracture of the tip of the petrous portion of the temporal bone, as he is of the opinion that a complete division of the nerve would not have recovered suddenly. He thinks that a hæmatoma was formed which pressed upon the abducens and induced a temporary paralysis, and that this disappeared as soon as the clot was absorbed.

SCHOENBERG.

ROUSSEAU (54, **Paralysis of the oculomotor nerves during otitis media suppurativa**) enumerates the various types of oculomotor paralysis that may occur during acute and chronic otitis media purulenta and summarizes them in the following manner: (1) Paralysis of the sixth nerve, in benign cases, may appear in aseptic serous meningitis, or limited osteitis of the tip of the petrous portion of the temporal bone. In malignant cases it may appear in abscess of the cerebellum, or extradural abscess at the apex of the petrous portion of the temporal bone. (2) Paralysis of the sixth and fifth nerves, the symptoms of Gradenigo, may be produced in benign cases by a slight osteitis of the apex; in malignant cases by an extradural

abscess, or localized meningitis. (3) Paralysis of the sixth and third nerves appears usually in thrombophlebitis of the cavernous sinus. (4) Paralysis of the third nerve in an abscess of the temporo-sphenoidal lobe. SCHOENBERG.

CAMILLE and FROMAGET (50, **Latent nystagmus**) again call our attention to a condition they have already described under the term "latent nystagmus" and give five more histories of such cases. The condition seems to be analogous to latent strabismus. They believe that just as there is a fusion center which prevents heterophoria from becoming manifest, there is a center, located in the cortex, which keeps the nystagmus in a latent condition. As soon as one of the eyes is excluded from vision the function of this center is interfered with and the nystagmus appears. In each of the five cases reported the patient had one bad eye, and when the good one was screened a nystagmus appeared in the other. SCHOENBERG.

MENACHO (53, **Associated movements of the upper lid during mastication**) reports a case in which the right upper lid was retracted every time the lower jaw moved downwards. He discusses the various possibilities of nuclear, subcortical, and cortical localization, as well as of an abnormal arrangement of the connecting fibers between the nuclei of the fifth and seventh nerves, and declares his inability to agree with any of the theories which have yet been advanced.

SCHOENBERG.

SUKER (55, **New ocular muscle symptom in exophthalmic goiter**) asserts that, after extreme lateral rotation of the eyes, when an attempt is made to rapidly fix an object in the median line, one of the eyes fails to follow the other or lags behind, but eventually jumps into place with the other.

ALLING.

A few attempts have been made in general surgery to transfer an active muscle to take up the action of a paralyzed one. WOODRUFF (56, **Tendon transplantation of the eye muscles**), following the technique of Hammelsheim, has operated on two cases of paralysis of the external rectus by dividing the superior and inferior recti tendons and suturing half of each to the tendon of the externus. The results seem to have been a slight improvement in the internal strabismus, but no restoration of motility outward. ALLING.

V.—**Tests for Color Blindness.** By Professor SHINOBU ISHIHARA, Tokyo Handaya, 1917.

This series of plates is designed to discover quickly defects in color perception, particularly in railway employees, candidates for the navy, and others. The color effect which enters into consideration is always congenital and concerns red and green blindness. The plates are so designed as to apply the peculiarity of the red and green blind consisting in the fact that blue and yellow colors appear to them much brighter than the red and green colors. Each plate consists of a collection of round disks of pigment of varying diameter in which a central figure against an appropriate background will appear as a certain number to the normal person and as a different number to the abnormal individual. The tests are very ingeniously designed and undoubtedly furnish a valuable addition to our equipment for detecting color defects. The get-up of this little book is worthy of note and is a testimonial of the high standard of bookmaking in Japan.

A. K.

VI.—**Oral Roentgenology.** By Dr. KURT H. THOMA, Lectures on Oral Histology, Harvard Univ. Dental School. Pp. 213. 311 illustrations. Boston, Ritter & Co., 1917.

The importance of dental infection in eye disease requires some knowledge of the X-ray findings in normal and pathological tooth conditions. This book, a Roentgen study of the anatomy and pathology of the oral cavity, admirably furnishes this information. The normal oral tissues and their pathological changes are carefully described and illustrated by Roentgenograms. The author, however, correctly states that

the Roentgen examination does not replace all other means of diagnosis, but it should be used in conjunction with other well-recognized modes of examination. The book is a scientific contribution to an important subject and its practical value is increased by brief case reports and their X-ray findings.

A. K.

VII.—**Military Ophthalmic Surgery.** By Drs. ALLEN GREENWOOD, GEO. E. DE SCHWEINITZ, and WALTER R. PARKER. War Manual No. 3, pp. 115, illustrated. Lea & Fibiger, Phila. and New York, 1917. Price $1.50.

This little book is designed for medical army officers who have to deal with special ophthalmic problems. The first part on Military Ophthalmic Surgery is written by Greenwood and is based on the results of experience gained at the war front in the British hospitals. This describes contusions and wounds of the eyeball, the implantation of a glass ball after enucleation, the value of the X-ray in localizing foreign bodies, the methods for their extraction, the use of conjunctival flaps, traumatic cataracts, wounds of the orbit and of the eyelids, and a brief reference to the fundus changes in trench nephritis and intracranial injuries. Finally, a list of the eye instruments for a base hospital is given. Trachoma and the Common Forms of Conjunctivitis are described by de Schweinitz, and Parker contributes a chapter on Malingering.

A. K.

VIII.—**The Stereoscope in Ophthalmology.** By Dr. DAVID W. WELLS, Boston, pp. 143. Published by Globe Optical Co., Boston, 1918.

The little book which appeared in part in 1912 deals principally with stereoscopic fusion training. The author recommends the phoro-optometer for this purpose which makes use of the principle of the insinuation of the prismatic element by decentering strong spheres, the objects remaining fixed. The stereoscopic treatment of exophoria, esophoria, hyperphoria, and heterotropia is then taken up in turn. A chapter is devoted to the musculo-capsular advancement operation and the author gives his modification of the Worth technic,

consisting in a so-called scleral anchor and the use of a fixation hook. Finally the Haitz method for the binocular examination for scotomata with the stereoscope is fully described; in this test the author prefers to use the phorooptometer.

A. K.